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An Address.[1]

By J. H. STUBBE,
Perth, Western Australia.

THE preparation of a valedictory presidential address has been my sword of Damocles for the last six months; each busy day it has sunk closer towards the nape of my neck. You will realize that each of the six Branches of the British Medical Association in Australia fondly expects the production of a well-considered and polished address by its retiring or incoming president each year. There is a dearth of apposite medical subjects for such addresses; consequently these speeches usually overlap in content and so lack originality, and most degenerate, as will mine, into a haphazard commentary on parochial matters, educational, ethical or medico-political.

My obvious opening gambit is to discuss the work of our local Council and its myriad committees and subcommittees. I have now been on this Council for four years, and it appears to me that the volume and scope of its task have been increased by 50% in each of those years. By every mail the Federal Council asks our Branch's opinion on knotty matters, particularly those pertaining to the working of the health services. There is certainly here in Western Australia every justification for the appointment of a capable and vigorous full-time medical secretary, such as the Victorian Branch has. However, because of our small numbers and the upkeep of our medical library, ours is a very expensive Branch to operate; our membership subscription is already the highest in the Commonwealth. Thus the work falls heavily on the time and grey matter of the few who are repeatedly pressed into service. And once one is on the Council, in spite of lengthening teeth and whitening hairs, the arguments of "continuity of service" and "*noblesse oblige*" make it almost impossible to fade away decently. My present plea is at least for more cooperation and less grizzling from members who themselves would not be sufficiently zealous to accept nomination.

Health Services.

I repeat that probably two-thirds of our time has been preoccupied by various facets of the National Health service. After many vicissitudes Sir Earle Page's scheme is well and truly launched, on a fairly even keel, and almost certainly unsinkable. Such a large proportion of the population is now insured against medical and hospital expenses that no future Federal Government would attempt to capsize the scheme, though amendments and alterations in its navigation will undoubtedly be sought.

The benefit societies now have better trained staffs who cooperate with doctors and patients much more closely than they did. We see fewer attempts at "direct payment",

[1] President's address, read on behalf of Dr. Stubbe by Dr. A. L. Dawkins at the annual meeting of the Western Australian Branch of the British Medical Association on March 19, 1955.

and "hardship forms" are being more properly used. Doctors are becoming more inured to the irksome clerical work, and the few brigands in our profession are being disciplined quietly, but often firmly. Those thus inclined to free-booting should remember that, whereas once the doctor's fee was a more or less private matter between him and his patient, it is now open to observation by the staff of the insuring societies, who frequently report what they consider excessive charges to the Health Department and to the Fees Advisory Committee of the local Branch of the British Medical Association. The superchargers are thus widely criticized by a section of the public and by some of their confrères.

The Honorary System.

Another question which has occupied much time is that of the medical staffing of government hospitals and the classification of hospital patients. Surely the honorary system is doomed. Can you visualize other professions giving long hours of free service to the government? There is now little need for philanthropy or charity from the medical profession. A sessional system of payment of a part-time visiting staff is being gradually introduced in several public hospitals in the eastern States.

In our own public hospitals a large proportion of the patients are insured against medical expenses; yet the hospital-employed assessors will not take such insurance into consideration at all when deciding whether a patient is to be admitted to a public or private bed. The anomaly then arises that the provident patient who has insured against medical expenses may be treated in a public bed by an honorary doctor; his premiums have been paid for nothing and only the insuring body benefits. Here is cause for thought and for early combined action.

Antibiotics.

We still discuss and advise against the overuse and misuse of expensive antibiotics in minor illnesses. These drugs have proved life-savers and should be guarded as such. Does one sabotage one's lifebelt? Diseases which were lethal have been controlled or wiped out. Sepsis and venereal disease are almost eliminated; but we are being notified daily in the medical Press of the appearance of more antibiotic-resistant infections. Recently our own maternity hospitals were cursed with a protracted epidemic of staphylococcal infections of infants and of maternal breasts, which was resistant to all the antibiotics. Yet many practitioners, much influenced by showers of blotting-paper advertisements, still do a great disservice to their patients by prescribing these drugs for conditions which are easily amenable to treatment by other methods. We must get out of the easily acquired habit of initially using these drugs for mild infections and for prophylaxis against sepsis.

The Treatment of Natives.

The matter of some State Government compensation to country doctors for the medical treatment of natives has been vigorously pursued. At first Cabinet refused to take any action, but we persisted and at a recent conference officers of the Department of Health and the Department of Native Affairs admitted our claims. The portents are favourable for the introduction of some reasonable rate of remuneration.

Good Manners.

Now I will embark on a delicate controversial subject. I am aware that my criticism may be repudiated by some of the audience. But a retiring president has the right of gentle castigation, and I recollect that I shall be 12,000 miles away at the time when this address is delivered.

We have been much perturbed by several complaints of neglect and rudeness against practitioners. It is agreed that the overworked doctor will at times be severely tried and frustrated by unnecessary and ill-timed demands for attention—often for minor or imaginary illness. But when a doctor sets up in practice he virtually guarantees to attend faithfully to his clientele; he becomes their "very present help in trouble", their ultimate authority in sickness. He should look at their distresses from their point of view and always regard them as frightened human beings—not merely as instances of disease.

Let us be honest with ourselves. In recent years peevishness and lack of courtesy are conspicuous throughout the community. One meets it on the bus, in the shop, in the bank, on the roadway. Civility and honest service are rare. The old-time friendly relationship between doctor and patient has depreciated. Families are no longer devoted to their doctor; he is no longer their oracle; he does not arouse their loyalty. And why? Probably because in this age of materialism everybody is too harassed to secrete the milk of human kindness. The doctor is in too much of a hurry to listen patiently to protracted tales of woe or to attempt to explain away fears and phobias. He becomes brief and brusque, often high-handed and irritable; he must go on to the next case. His patient is left not understanding, and forlorn.

We must all try to solve this problem. It is our duty, and should be our pleasure, to attempt to ameliorate human unhappiness by kind attention to all. If some of our patients are utter nuisances, we should do our best to conceal our feelings about them. This is a counsel of perfection, I admit; but it will pay dividends in the long run.

Drunken Driving.

We have been in liaison with the Law Society on the questions of drunken driving and road safety. The toll of road accidents is far too heavy. The general public must be taught that they should not drive a vehicle when their skill or judgement is distorted by alcohol. After a few drinks a driver may feel that he is driving magnificently, but his critical faculties and nicety of judgement are in fact impaired; he is apt to take risks which he would never accept at other times. His degree of intoxication is perhaps not sufficient to attract the attention of the police or the onlookers; but it is sufficient to interfere with the instantaneous reactions of steering, braking or accelerating, which so often avoid catastrophe. How many of us, comparative sobersides, have after an infrequent celebration ("off duty") said to ourselves next morning: "What a fool I was, and how lucky to get home without scathe!"

In some countries it is possible for the police to enforce blood and urine alcohol estimations on any driver at any time, whether he has been involved in an accident or not. When, perhaps, some such plan has been evolved, we hope, in conjunction with the legal profession, to make a strong recommendation to the Government in an effort to mitigate the increasing motor-vehicle accident list.

Liaison with the Press.

We have achieved a very good understanding with the Press, who are continually referring to our Press liaison officer on medical subjects. His prepared statements, after consultation or reference to specialists when indicated, have to my mind been eminently clear, dignified and convincing to the public. They correct much erroneous and deceptive information submitted by well-intentioned reporters.

General Practitioners and Specialists.

I myself have seen little of the suggested friction between general practitioners and specialists; but I would strongly urge all specialists to be most meticulous in reporting fully and promptly on problems referred to them. It is admitted that this is an onerous duty, which must often be accomplished in overtime hours; but it should be remembered that the general practitioner and the patient are perhaps anxiously awaiting the opinion. For this purpose a recording machine and a competent secretary are initially expensive, but ultimately a conservation of time, energy and patience.

Finance.

With regard to our property in King's Park Road, the trustees have recommended that for the present we do not instal our offices there. Dr. A. L. Dawkins will clarify the financial position for you. It is hoped that soon our equity

in the property will be increased by the acquisition of a substantial donation from the Protection of Practices Society.

A Medical School in Western Australia.

The project of a medical school within the University of Western Australia has never met with opposition, but there has been much apathy to overcome. Your Council has been nagging at all and sundry for years without result. I will only briefly recapitulate some of the many cogent reasons for the establishment of a medical school; these include the raising of the standard of medical practice and of the doctor ratio in our rapidly increasing population; the hurdle to aspiring students of prolonged and expensive deportation to another State; the overcrowding of the other schools which at present bear the unwanted burden of undergraduate and post-graduate training of Western Australian students; and the inherent loss to health, hospital, scientific and industrial research organizations in this State. When we heard that nothing concrete had eventuated after a comprehensive report from the University had been submitted to the Premier, we decided to try the effect of a personal talk with him. Our deputation carefully avoided any political side issues. The Premier promised to find half the capital cost and all the maintenance. We, the Council of this Branch of the British Medical Association, have undertaken to give the school our whole-hearted support; specifically, we have promised the use of our library, any teaching we are asked to contribute, and some measure of financial support. We have also engaged to use our influence to induce the Commonwealth to lend a hand. So far, so good; but we must not be content. The ball is not now in our court, but we must strive to see that it is kept in play until the game is finally won.

To turn to the organization of the school, my thoughts are probably not at all original; and as this speech was drafted in late January, more recent pronouncements from the University or its advisers may have made my suggestions redundant. The curriculum must, of course, be approved by the General Medical Council, to ensure reciprocity of registration for our graduates with all British Commonwealth countries. The staff must be headed by a full-time outstanding authority on medical education as Dean of the Faculty; around him must be gathered the best teachers available — whether full-time, part-time or honorary. I believe the Faculty should be strongly represented on a permanent post-graduate committee, so that the teaching of students, resident medical officers, general practitioners and specialists should be continuous, consecutive and progressive.

Because of the dearth of finance, the pre-clinical departments must originally be ignobly accommodated. Most Australian medical schools were born in tin shanties. We should require at first merely shelter for classes and equipment; two-thirds of such accommodation, I understand, already exists. But scholastically, these departments must be first-grade.

The clinical training should give little trouble; we have well-qualified clinicians, and there is an unexploited wealth of clinical material in our existing public hospitals. Admittedly, much of this material is in private beds and nominally at present unavailable for teaching purposes; but I believe there would be little difficulty in making it accessible to students. It could be a *sine qua non* for admission of any patient to a public hospital that he would not object to the doctor's using his disability for teaching and demonstration purposes.

Here, if I may diverge, I should like to air my belief that more emphasis should be laid on teaching students to be good general practitioners. Of our doctors, 85% are general practitioners, and they are usually the first contact and defence against the attack of disease. They, particularly in outback areas, must make the provisional diagnosis and initiate treatment; obviously much depends on their powers of observation and knowledge of correct treatment at the onset of all types of illness. A general practitioner was once asked by a stranger what his specialty was. He replied: "The skin and its contents." It is a fact that most medical teachers, salaried or honorary, in schools, hospitals, or post-graduate courses, are specialists absorbed in their own particular subjects and unacquainted with the common minor ailments. Many have never treated patients in their own homes, and have no insight into the effect of domestic background conditions on physical or mental health. Some specialists never realize that this man's father's death from a stroke, or this woman's mother's lingering illness from a uterine carcinoma has much to do with the present patient's illness. Their patients are not their friends, and they are unable to gain their complete confidence. Here is where an experienced general practitioner has much to teach the student on the practical management of patients. I would therefore advocate a lecturer on general practice and an extension of the recently introduced system by which students are attached to suitable suburban or country doctors as "observers" in the daily routine; thereby they might better obtain a thorough insight into the difficulties and manifold rewards of general practice. I would go further and suggest that it be mandatory before examination for specialist diploma or degree, that the candidate should have completed a period of general practice. I am informed that in South Africa this period is five years; probably two years would be adequate.

When our medical school opens its doors it is undoubted that there will be more applicants than can be admitted to the course, and some type of preselection will have to be enforced, to ensure that all entrants are of good quality. I imagine that a selection committee would personally interview all candidates. The first requisite would be a medical certificate of physical fitness.

The medical course and medical practice involve long and irregular working hours, and demand good physique and resistance to infections. The weakling student obviously wastes his parents' money, his teachers' time, and the school's accommodation. A doctor will, equally obviously, best serve the community if he remains well and lives long.

The selection committee would have confidential access to scholastic records and headmasters' reports. Whatever the proponent's view as to his ultimate medical goal—general practice, specialist practice, science, research *et cetera*—he should, on being interviewed, give indications of certain basic characteristics—ambition, personality, sound judgement, responsibility, honesty *et cetera*—and, if possible, some flair for social leadership. The young graduate going to a one-man country practice will almost certainly be elected chairman of the parents and citizens' association, chairman of the local hospital committee, a member of the roads board, perhaps (it is hoped) captain of the cricket team, and even a justice of the peace. In the city areas he will be in demand for marriage guidance, philanthropic and artistic committees *et cetera*. Thus it is really important that, to uphold our prestige, our doctors should show some social graces and the capacity to make a good speech. The applicant should be earnest in his desire to become a doctor. He should "have a call" to a noble profession and not be merely desirous of making a good income or "joining a good club".

Those applicants chosen might, after one probationary year and perhaps an examination, be reassessed, the Faculty having the power to expel them before more time and money are wasted if their performance was considered unsatisfactory.

After graduation at least one year should be served as resident medical officer at a recognized hospital before registration by the Medical Board is allowable. It is a fact that recently some young graduates could not obtain a hospital appointment; they were "loosed" on the public without experience or confidence, and considerable damage may have been done to their patients and to our good reputation with the public.

Having qualified, been duly registered, and taken up practice, the general practitioner in these days has little opportunity for improving his knowledge. Without a higher degree he has no hope of a hospital appointment, and little opportunity of clinically rubbing shoulders with

his confrères. I believe all general practitioners should somehow have access to hospital beds and clinical teaching; a doctor without a hospital appointment is at a dead end and loses the spur to excel. Why should not eager young general practitioners be appointed, as they desire, to deputy assistant clinical assistantships or some such positions on the staffs of the public hospitals?

The general practitioner can, and does, attend some post-graduate courses; but how many of the lectures he hears and the demonstrations he watches help him in his every-day work? Such courses should be more designed to illustrate the most modern methods of diagnosis and treatment of everyday complaints rather than to discuss abstruse and rare cases more suitable for argument amongst members of the Royal Colleges. Let the lecturer remember that some of his audience examine their patients on a sagging double bed in a poorly lit room, without ready laboratory or consultant facilities, and that they must reach and pronounce a firm conclusion before they leave the home.

Without some hospital follow-up and clinical contacts our general practitioners are in danger, as happens in some other countries, of becoming mere pigeon-holing devices—only allocating their patients to this or that hospital or specialist, and learning little or nothing of the course and termination of their patients' illness.

Conclusion.

I must apologize to you for my physical absence this evening, some weeks before the end of my term of office. I must also apologize to Dr. Dawkins for thrusting upon him the duty of delivering my address. In this I am no exception to the general rule that doctors do not deliver their own conceptions. I desire to thank sincerely the fifteen Pillars of Wisdom who have so stoutly carried their share of the burden on the Council during 1954, and I thank, too, those non-councillors who have agreed to being coopted for arduous duties on subcommittees. I am indebted to Mr. Hugh Hancock and Miss Wendy Lunnon for competent and courteous secretarial duties. Finally, I assure you that although I have been ever conscious of my shortcomings as a chairman and speechmaker, I have been intensely proud of my accession to the presidency of this Branch of our Association in 1954-1955.

ANEURYSM OF THE SPLENIC ARTERY AND CHRONIC PANCREATITIS, WITH A REPORT OF SUCCESSFUL SURGICAL RESECTION.

By E. S. R. HUGHES AND R. A. JOSKE.[1]

From the Royal Melbourne Hospital and the Clinical Research Unit of the Walter and Eliza Hall Institute, Melbourne.

ANEURYSM of the splenic artery is an unusual though recognized cause of upper abdominal pain and hæmorrhage. Sherlock and Learmonth in 1942 reported 119 cases from the literature and five more of their own, and Tonge, in 1948, in his excellent review in this journal collected 145 cases. Since then occasional further case reports have appeared (Sheehan and Falkiner, 1948; Taggart, 1952). In 1953 one of us (E.S.R.H.) operated on a patient with spontaneous rupture of a splenic arterial aneurysm, but apart from this case, no others have been encountered by us.

Ætiology.

Splenic artery aneurysm usually results from atheroma or from embolism, but other rare causes have been reported, such as congenital defects, trauma, gastric ulcer, gastric neoplasm, portal hypertension, syphilis, Gaucher's disease and splenic arterial thrombosis. An unexplained fact is the relative frequency of splenic arterial aneurysm in pregnancy; approximately 20% of the reported cases have occurred in pregnant women (Lennie and Sheehan, 1942; Sherlock and Learmonth, 1942; Cosgrove *et alii*, 1947; Sheehan and Falkiner, 1948; Chalmers, 1949; Gallagher and Hudson, 1954).

In the case to be presented in this paper the association of the aneurysm with chronic pancreatitis is of interest, since Leger and his associates (Leger *et alii*, 1954) have recently emphasized the relation between splenic and pancreatic lesions, and the association of vascular disease and pancreatitis has been stressed by several workers (Burn, 1951; Heinz, 1952; Hranilovitch and Baggenstoss, 1953; Saint, 1954).

Surgical Pathology.

In some cases the splenic artery ruptures without aneurysm formation (Macleod and Maurice, 1940; Gillam, 1942). Occasionally an arteriovenous aneurysm forms, either between the splenic artery and the splenic vein or within the spleen itself (Evans, 1954). In the case reported here the aneurysm was 10 centimetres in diameter and had thick fibrous walls and laminated clot in the centre. It appeared to arise from the main splenic artery and involved the pancreatic body.

Clinical Features.

Atypical left upper abdominal pain is a frequent symptom of splenic artery aneurysm. A mass may be palpable in the abdomen, as in this case. It is usually on the left side under the costal margin, and may be pulsatile and associated with an audible bruit. Radiologically the aneurysm may be responsible for a pulsating deformity of the stomach (Sperling, 1940), or its walls may be calcified. If rupture occurs, the symptoms and signs are those of massive intraabdominal hæmorrhage. During pregnancy this may simulate a ruptured ectopic gestation, or if it occurs later in pregnancy ,concealed accidental hæmorrhage Rupture usually occurs into the lesser sac, and rarely into the general peritoneal cavity.

The site of the hæmatemesis which occurred in the present case remains uncertain. No peptic ulceration was detected radiologically, by gastroscopic examination or at operation; but the aneurysm was adherent to the stomach, and it is presumed that an acute gastric erosion occurred in this area.

Treatment.

The treatment is excision of the aneurysm and splenectomy. When removal is not practicable, proximal and distal ligation of the splenic artery has been practised (Marshall, 1922; Parsons, 1936). In the case reported here a large aneurysm was successfully resected, and it was necessary to remove the spleen and most of the pancreas to accomplish the procedure. The extent of the pancreatectomy can be gauged by the development of *diabetes mellitus* in the post-operative period, although the associated chronic pancreatitis also contributed to this. A similar case, with a similar successful resection, has recently been reported (Williams and Harris, 1954).

In the present case an aneurysm of the splenic artery, responsible for upper abdominal pain, could be palpated as a discrete tumour under the left costal margin. Laparotomy revealed a mass in the body and distal half of the pancreas, which was thought to be a carcinoma. The mass was excised, together with the spleen and most of the pancreas. Pathological examination revealed the mass to be an aneurysm of the splenic artery. Post-operatively the patient developed *diabetes mellitus*, but otherwise his convalescence was satisfactory.

Report of a Case.

A male patient, aged forty-eight years, was admitted to the Royal Melbourne Hospital on September 29, 1954, and was discharged on November 4. Five years previously he had developed epigastric pain radiating through to the back and up to the left shoulder. This pain was severe and unaffected by either food or alkaline powders. It occurred at frequent intervals with short periods of relief, though he had never been free of pain for longer than a month. For two months before his admission to hospital this pain was accompanied by vomiting. A week before his admission he vomited some bright blood and also noted some blood in his

[1] Working with the aid of a grant from the National Health and Medical Research Council of Australia.

motions; for two days prior to his admission he had vomited "blackish material" at frequent intervals. His only previous illness was an operation for hæmorrhoids in June, 1952; at that time routine examination failed to disclose an abdominal mass.

On examination the patient was slightly febrile (temperature 99° F.) and appeared pale. His blood pressure was 160 millimetres of mercury, systolic, and 100 millimetres of mercury, diastolic. A large, hard mass was palpable in the upper left quadrant of his abdomen, extending from under the left costal margin towards the umbilicus and measuring about 10 centimetres in diameter. It was not tender; it was pulsatile, but the pulsations appeared transmitted. No other abnormality was discovered on physical examination, and there was no evidence of vascular disease.

A provisional diagnosis of gastric carcinoma or of aneurysm of the aorta was made.

A number of investigations were carried out with the following results. The hæmoglobin value on his admission to hospital was 6·6 grammes *per centum*, rising after blood transfusion to 10·3 grammes *per centum*; the white cell count was 5000 per cubic millimetre. The cephalin flocculation test produced a positive result. The serum albumin content was 3·9 grammes *per centum*, the serum globulin content 1·9 grammes *per centum*, and the serum bilirubin content 0·5 milligramme per 100 millilitres. Occult blood was present in the fæces. X-ray examination with a barium meal showed the stomach to be normal, but the duodenal cap asymmetrical. The Wassermann test produced a negative result. An electrocardiogram was within normal limits. Dr. J. L. Stubbe made a gastroscopic examination and reported as follows: "The stomach was difficult to distend with air. No ulceration was seen. The stomach was pushed in posteriorly on the lesser curvature above the angulus. This suggested either extrinsic pressure or a non-ulcerating gastric neoplasm."

Because of this diagnostic uncertainty, and because the patient had a further hæmatemesis while in hospital, laparotomy was performed on October 22.

The abdomen was opened through an upper right paramedian incision. A rounded discrete mass, 10 centimetres in diameter, was found anterior to the aorta and extending to the left towards the spleen. It projected into the lesser sac above and behind the body of the stomach. It was intimately related to the pancreas, and, indeed, seemed to have replaced the body and tail. It was smooth and firm, with pulsations transmitted from the closely related aorta. It was slightly mobile up and down, but not sideways. Further exploration revealed the spleen to be slightly enlarged and the posterior wall of the stomach adherent to the whole of the anterior aspect of the tumour. The head of the pancreas was firm and a little enlarged. No free fluid was present and no other abnormality could be detected within the abdomen.

The lesser sac was opened above and below the stomach. The spleen, splenic vessels and tail of the pancreas were successively mobilized. As the dissection proceeded to the right, it became clear that the tumour could be excised only by the removal of portion of the pancreas. Some difficulty was experienced in recognizing the branches of the cœliac artery because of the proximity of the edge of the tumour. The superior mesenteric vessels were identified and freed, and the pancreas was divided on their right and immediately adjacent to the common bile duct (Figure I). This left less than one-quarter of the pancreas behind. The pancreatic ducts were oversewn. The posterior wall of the stomach was excised with the tumour, and the resulting long defect was repaired in two layers. The operation was completed in three and a quarter hours, and the patient's condition was good at the conclusion of the procedure.

His immediate post-operative course was uneventful; there was no drainage from the abdomen, and bowel sounds returned promptly. His mid-morning blood sugar level on October 25 was 170 milligrammes per 100 millilitres. He later developed a wound infection, but was discharged to a convalescent hospital on November 4. There was still some discharge from the wound at this stage.

In the week before Christmas he became very thirsty. This thirst persisted despite the ingestion of large quantities of fluid which was associated with the passage of considerable amounts of urine at frequent intervals. He felt weak and began to lose weight, and was therefore transferred back to this hospital on January 7, 1955, when his fasting blood sugar level was found to be 390 milligrammes per 100 millilitres. A glucose tolerance test on January 13 revealed a typical diabetic curve. His condition was stabilized on a daily dose of 12 units of NPH insulin, and he was discharged from hospital on January 29. When last examined on February 21 he was well and gaining weight. His diabetes was now controlled by diet alone, without the administration of insulin.

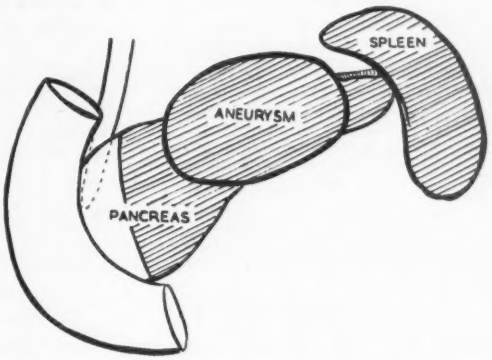

FIGURE I.

Sketch showing the extent of surgical resection. The aneurysm, the spleen and the greater portion of the pancreas were removed.

Pathological Report on the Operative Specimen.

The specimen comprised the spleen, aneurysm, and portions of the pancreas and stomach (Figure II). The spleen measured 10 centimetres in diameter. The splenic artery was dilated and tortuous, and attached to it was a large, spherical, thick-walled aneurysm, eight centimetres in diameter, containing a small amount of laminar thrombus and much recent clot.

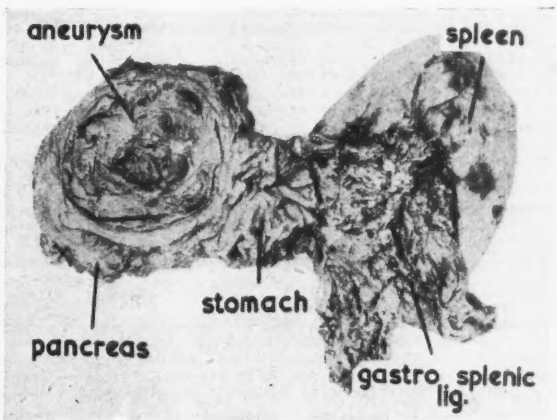

FIGURE II.

The operative specimen after dissection. The aneurysm has been sectioned and is seen to be filled with laminated blood clot. It measured eight centimetres in diameter.

Histological examination of sections taken through the wall of the stomach revealed relatively normal mucosa and muscle coat, with dense fibrosis and round-cell infiltration of the adventitia extending to the wall of the aneurysmal cavity, where there was a mass of blood clot. Examination of sections through the outer edge revealed organization of the clot from the edge by fibroblasts growing into it. Examination of the pancreatic tissue included in the sections revealed chronic pancreatitis with considerable atrophy of the acini, and infiltration by inflammatory cells (Figure III). It will be recalled that at operation the head of the pancreas was felt to be thickened.

Summary.

1. The incidence, ætiology, pathology, clinical features and treatment of splenic artery aneurysm are reviewed.

2. A case is reported of a large splenic artery aneurysm associated with chronic pancreatitis and treated by resection. The size of the aneurysm necessitated removal of considerable pancreatic tissue, and this with the associated chronic pancreatitis resulted in the development of *diabetes mellitus*.

Acknowledgements.

We are grateful to Dr. J. D. Hicks, pathologist, Royal Melbourne Hospital, for the report on the operative specimen. The photomicrograph was prepared by Mr. E. Matthaei, University of Melbourne, and the other figures by Mr. R. Ingles, clinical photographer, Royal Melbourne Hospital.

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Legends to Illustrations.

Figure III.—Photomicrograph of portion of the pancreatic tissue removed at operation. This shows the changes of chronic pancreatitis, fibrosis, cellular infiltration and atrophy of glandular elements. (Hæmatoxylin and eosin stain, × 25.)

SUBPHRENIC ABSCESS.

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Subphrenic infection is a not uncommon occurrence, which may progress to abscess formation. Hidden beneath a rigid concavity of ribs, such an abscess can be of very insidious nature and grow to a huge size whilst still escaping the attention of the clinician, who meanwhile searches in other channels for his diagnosis. However, should he maintain a mental awareness of its possibility, the very paucity of its clinical features will quickly act as a pointer and direct his attention to this hidden area.

Historical Review.

The first classical description of subphrenic abscess was made by Barlow in 1845, in the *London Gazette*, and this holds to the present day. The next step forward was taken by Barnard (1908), who, in a review of 76 cases, classified clearly for the first time the subphrenic spaces, and pointed out the individual characteristics of infection in each, and described the operative treatment. However, mortality in cases of proven abscess still remained high, until Nather and Ochsner in 1923 and Clairmont in 1926 pointed out the dangers of involvement of an uninfected pleural cavity, and respectively described the posterior and anterior extraperitoneal approaches to the abscess cavity. It remained for Harley in 1949 to clarify the position still further and give a detailed description of the radiographic appearances.

Material.

This paper is based on a review of the literature and on a study of 100 cases of subphrenic abscess which occurred in the civilian general hospitals of the Brisbane area during the period from 1930 to 1954. There were 70 male and 30 female patients.

The Anatomy of the Subphrenic Region.

The subphrenic region lies between the diaphragm above and the transverse colon and mesocolon below. This large space is subdivided by the liver into suprahepatic and infrahepatic compartments. The suprahepatic compartment is divided into right and left portions by the falciform ligament.

Barnard's classification of the subphrenic region remains in active use today. However, there has been much confusion by various authors regarding the various spaces. The terminology of the spaces in this paper is based mainly on that used by Harley (1949) and first postulated by Mitchell (1940).

There are six supracolic spaces, five of which are intraperitoneal and one extraperitoneal. The latter, lying between the bare area of the liver and the diaphragm, is not relevant to this paper.

The Intraperitoneal Spaces.

The following are the intraperitoneal spaces: (i) the right suprahepatic space, (ii) the right subhepatic space, (iii) the left suprahepatic space, (iv) (*a*) the left anterior subhepatic space, and (*b*) the left posterior subhepatic space.

The Right Suprahepatic Space.

The right suprahepatic space is situated between the diaphragm and the superior and anterior surfaces of the right lobe of the liver. Posteriorly, it is limited by the superior layer of the coronary ligament, which separates it from the extraperitoneal compartment, and around the lateral edge of which it communicates with the right subhepatic space.

The Right Subhepatic Space.

The right subhepatic space lies below the anterior edge of the liver and extends up behind the liver as Morison's pouch. It is bounded above and in front by the inferior surface of the right lobe of the liver and the gall-bladder, and behind and below by part of the right kidney, the structures lying on the anterior surface of the latter, and the transverse mesocolon. Superiorly, it projects upwards in the form of a recess, between the liver in front and the kidney, suprarenal gland and *vena cava* behind. It is limited above by the inferior layer of the right coronary ligament, and below it opens into the right lateral paracolic gutter. Its recess is the lowest part of the right side of the abdomen proper with the body in the supine position, and so effusions tend to gravitate there. To the left its space is limited by structures in the free edge of the gastrohepatic omentum.

It has long been taken for granted that there are two suprahepatic spaces on the right side. This was Barnard's hypothesis, and has been supported by McGregor (1932), by Ochsner and Graves (1933) and by Thorek (1947).

However, as Mitchell clearly points out, it is easily seen in the living body, and also in the cadaver, that the posterior suprahepatic space is really Morison's pouch—a postero-superior cul-de-sac of the right subhepatic space (Figure I).

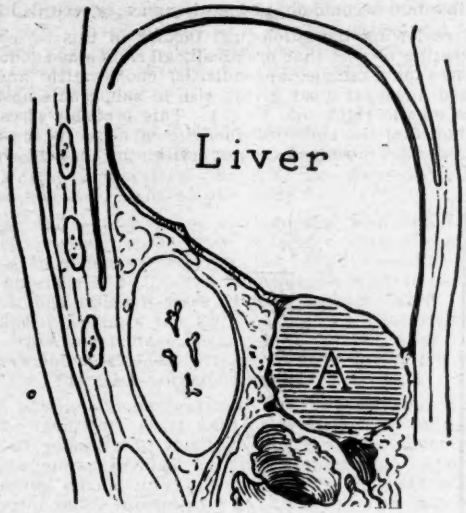

FIGURE I.
Anatomy of the right subphrenic region. (From Ochsner and Graves, 1933, by permission.)

The Left Suprahepatic Space.

The left suprahepatic space is a potential cavity beneath the diaphragm, and above the anterior and superior surface

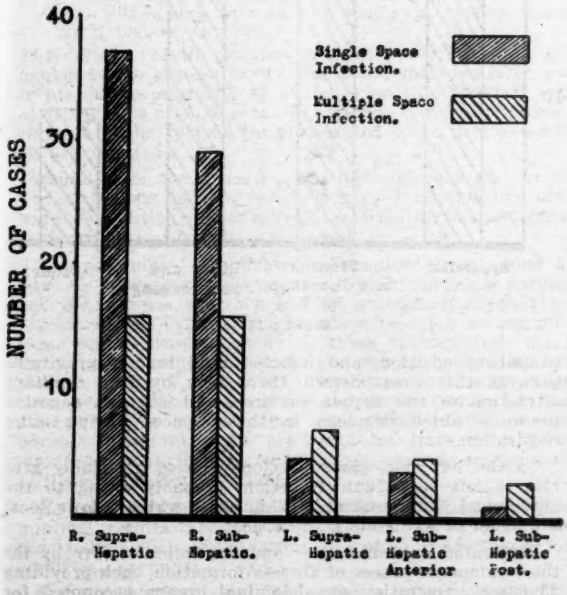

FIGURE II.

of the left lobe of the liver, the stomach and the spleen. It is separated from the right suprahepatic space by the falciform ligament. Posteriorly, it is limited by the superior layer of the triangular ligament and is separated from the lesser sac by the lieno-renal ligament. The extent of this space and its communication with other spaces depends to a large extent on the size of the left lobe of the liver.

The Left Subhepatic Space.

The left subhepatic space has been divided into the anterior and posterior spaces. The left anterior subhepatic space lies below the left lobe of the liver in front of the gastro-hepatic omentum. It can communicate fairly easily with the left suprahepatic space by the interval between the liver and stomach. The left posterior subhepatic space is the lesser sac of the peritoneum. It varies considerably in extent. In time of infection its communication with the right subhepatic space, the epiploic foramen, readily becomes sealed off.

Reference to Figure II will reveal the frequency with which the various intraperitoneal spaces were involved, both in single and in multiple space infections.

INCIDENCE OF SUBPHRENIC INFECTION.

In the present series of 100 cases the right side was involved alone in 80 cases, the left was involved in 14, and in five cases the infection was bilateral. Multiple spaces were involved in 25 cases. Of these latter, in 20 the infection occurred on one side, and in five it was bilateral.

Right Subphrenic Abscess.

As can be seen from Table I, the right side is involved far more commonly than the left. This can be accounted for easily by the frequency of intraperitoneal infections, especially cholecystitis, perforated duodenal ulcer, and appendicitis. These three conditions occurred on 51 occasions, and involved the right side only in 100% of cases.

TABLE I.
Side Incidence of Subphrenic Infection (100 Cases).

Side.	Number of Cases.
Right	80
Left	14
Both sides	5
Side not stated	1

Because of its relative frequency, infection on the right side often takes attention away from the left side with, in consequence, the occasional overlooking of the abscess and a fatal issue. Also of some importance is the relative rarity of a neoplastic cause for the right-sided lesion, so that the patient's tissue resistance is greater. There were only two carcinomata, both colonic, causing abscesses on this side in this series. As will be seen, carcinoma has a much more sinister part to play on the left side. It is because of this awareness of right-sided abscess and its greater ease of diagnosis that its mortality is lower. In the present series there were 80 single and multiple spaces infected on the right side, with 20 deaths, a 25% mortality.

Left Subphrenic Abscess.

The left side was involved alone in 14 cases, and in conjunction with the right side in five cases. Multiple spaces on the left side alone were involved in 14 cases. Therefore subphrenic abscess on the left side is a relatively common occurrence.

To many clinicians subphrenic abscess means right subphrenic abscess; but it is important to bear in mind the high incidence of left-sided abscess and the possibility of infection on this side if the patient's condition does not improve after right-sided drainage.

Left-sided abscesses are more difficult to diagnose because of the following features.

The left subphrenic space is bounded by mobile structures such as the spleen, the stomach and the left lobe of

the liver, and the abscess tends to be multilocular and subsequent drainage inadequate. The diaphragm on this side rises less, so that intrathoracic changes are also much less common. The slight elevation of the diaphragm on the left side is not noticed so much as the same amount of elevation is noticed on the right side, because of the equalization in heights. For the same reason immobility of the diaphragm is less common. Neuhof and Schlossmann (1942) demonstrated an elevated paralysed diaphragm in only 60% of cases of left-sided abscess. Also the left subphrenic gas bubble may be mistaken for a normal stomach bubble.

In the present series of 14 cases, seven patients died, the deaths being due in part to non-diagnosis, to the affection which was the primary cause of the abscess, and to inadequate drainage. The primary lesion is important because of the high incidence of carcinoma on the left side. Of the 14 cases, six were due to carcinoma of the stomach, and five of these patients died.

Multiple Space Infection.

The mortality of subphrenic abscess varies directly as the extent of the infection and its unilateral or bilateral nature; single abscesses in this series had a mortality rate of 32%, the mortality rate of multiple unilateral space infections rose to 45%, and in the bilateral series the rate was 100%; that is, the combined mortality rate was 56%. Reference to Table II shows this interesting rise.

TABLE II.
Multiple Space Infections.

Type.	Number of Cases.	Number of Deaths.
Unilateral	20	9
Bilateral	5	5
Total	25	14

Of the nine patients with unilateral space infections who died, seven had no drainage, and of those that lived, all underwent drainage either once or twice. Obviously, the longer the abscess is left, the higher the mortality.

The 100% death rate in the cases of bilateral infection clearly indicates the necessity for the constant awareness of its possibility. There is nothing to be lost by exploration if an abscess is suspected on the other side after one drainage.

The main reasons for the deaths in the bilateral series were as follows: (i) the presence of general peritonitis in association with the abscess; this occurred on one occasion; (ii) the missing of an abscess; this occurred in three cases and may be attributed to the difficulty in diagnosis on the left side. In one case a large right-sided abscess was drained, but a left-sided abscess was missed, because of unequivocal clinical and radiological findings. In the other two cases infection on the right side only was suspected, but no drainage was carried out.

ÆTIOLOGY.

As a rule the source of infection is located in the abdomen and is the result of (*a*) an acute abdominal crisis, (*b*) operative interference giving rise to local or general peritonitis.

As can be seen from Figure III, the appendix, gall-bladder, stomach and duodenum were the sites most frequently involved.

Spread from outside the abdominal cavity, from the pleural cavity, is not recorded in this series. Harley (1949) found the pleural cavity to be the primary site in 5% of his cases. The reason for this low percentage is probably that the lymphatic flow is from the abdomen to the chest, and not *vice versa*. This would account for the high incidence of thoracic complications following subphrenic abscess. Other sources of subdiaphragmatic infection by hæmatogenous or lymphatic spread from distant foci, such as osteomyelitis or suppurative arthritis, are possible but uncommon, and no instance is recorded here.

In reviewing the ætiological factors in this series it is interesting to note that practically all right-sided abdominal lesions—for example, appendicitis, cholecystitis and perforated duodenal ulcer giving rise to subphrenic abscess—do so on the right side (97%). This probably rises from the fact that the right infrahepatic cul-de-sac is the lowest part of the abdomen proper with the patient in the

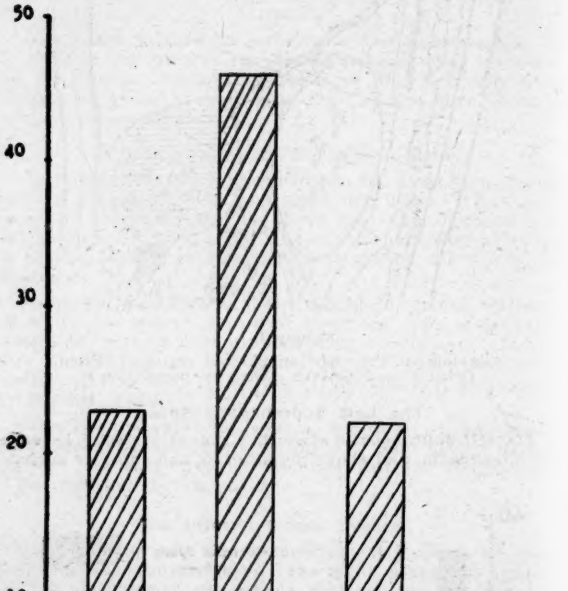

FIGURE III.

recumbent position, and infected fluid readily gravitates towards this area, drawn there also by (*a*) capillary attraction of two serous surfaces, and (*b*) the negative pressure which develops in these spaces during quiet respiration.

On the left side, gastric lesions almost invariably give rise to left subphrenic infection, probably owing to the anatomical configuration of this area, which allows local extension of fluid into these spaces.

Perforated duodenal ulcer and appendicitis were by far the commonest causes of abscess formation, each providing 23 cases. Operation on abdominal organs accounted for 24% of subphrenic abscesses. Gastrectomy was an ætiological feature in ten cases, and of these patients seven died, which is a very high proportion as compared with other causes.

It is to be noted that the mortality rate of subphrenic abscess associated with carcinoma of the stomach, whether as a result of operation or of spontaneous perforation, is

100%, which is probably accounted for by the poor healing powers of the stomach, the seat of neoplastic changes together with—and this applies to simple lesions also—the protein depletion consequent on prolonged dieting, and restriction of oral intake after operation.

Diseases of or operations on the liver or biliary passages accounted for 22% of all abscesses, which were invariably right-sided. Liver abscess as a primary ætiological factor occurred once only. The subphrenic abscess, of course, may extend and involve the liver secondarily in the infective process.

CLINICAL FEATURES.

Barnard summed up the clinical features very succinctly in his famous aphorism: "Signs of pus somewhere, signs of pus nowhere, signs of pus there."

After the operation, or concomitant with the inflammatory process, which may be under chemotherapeutic control, there is a regression in the patient's condition. He feels anorexic and ill. The temperature remains elevated, or if it had fallen it rises again without apparent cause, and finally assumes the swinging nature associated with pus. This unexplained fever which persists after a suppurative intraperitoneal process is very suggestive. The pulse rate increases correspondingly.

The patient sweats profusely and is prostrated. There may be transitory chest and upper abdominal pain, or a sense of pressure in the upper part of the abdomen or loins, often aggravated by deep respiration. A dry or productive cough may appear, and if there is a huge abscess or much intrapleural reaction, dyspnœa may occur (Nather and Ochsner, 1923).

On physical examination there may be no signs relevant to the subphrenic region. Careful palpation may demonstrate a tender spot, which increases daily, and eventually a mass may be evident. The skin may be reddened and œdematous, and there may be signs of pulmonary consolidation. The classical clinical picture was first described by Barlow (1845) as follows:

> Above the liver dullness is found, a tympanitic zone which is in turn covered by a zone of dullness produced by a pleural exudate.

This *bruit d'airain* results from the presence of a gas bubble in the abscess cavity, but as we shall see later, gas is present in only 32% of all cases, so this classical area of tympany is often absent. In only one case in the present series was the tympanitic area stated to be demonstrated on examination.

Jaundice is not a feature of subphrenic abscess *per se*. Three patients in this series developed icterus, but they were all fighting a generalized infection of the peritoneal cavity with a resulting toxic hepatitis.

The possibility of subphrenic infection always must be strongly suspected in every patient with continued pyrexia and leucocytosis who has had an antecedent suppurative intraperitoneal process, and in whom no other accountable focus can be demonstrated. If these characteristic manifestations of subphrenic infection persist instead of subsiding, within a period of a few days to a week, then the development of a subphrenic abscess must be considered. It is believed that approximately 70% of subphrenic infections which are diagnosed from the clinical manifestations subside without progressing to suppuration (Ochsner and Graves, 1933).

As can be seen, physical examination often produces minimal results, and the next step in the investigation, the radiological examination, is often undertaken on empirical grounds.

Sometimes, of course, the evidence of a collection is clearly discernible by the presence of an epigastric mass or the downward projection of the liver by the abscess. Especially may this be so on the right side, where the great bulk of the liver lies. However, usually there is no gross displacement of the liver downwards, which is in direct contrast to many cases of intrahepatic suppuration from which a subphrenic abscess has to be distinguished (Hailes, 1934). A left-sided abscess, surrounded as it is by yielding structures, is far less likely to produce an abdominal mass. In this series 22 epigastric masses were drained, and in only three instances was the abscess left-sided. Abdominal symptoms and signs only were present in 28% of cases.

Leucocytosis is almost invariably present. The average number of leucocytes in 54 cases in this series was 18,600 per cubic millimetre.

The presence of pneumonic changes is often detected; but the clinician must not be confused by the diminution in size of the pleural cavity caused by the elevation of the diaphragm by the abscess on its under surface. Symptoms and signs referable to the thorax only are more common with suprahepatic infections. Conversely, infrahepatic collections more commonly produce abdominal changes. However, the majority of abscesses are associated with both thoracic and abdominal changes. Harley (1949) found that 59% of abscesses fell into the latter category. In this series 54% involved thorax and abdomen.

Thoracic symptoms and signs only were present in 14% of single space lesions (Harley, 16%). These pulmonary changes are mostly sympathetic in nature, and consequently it is important to remember that pneumonic basal lung changes substantiate rather than hinder the diagnosis of subphrenic abscess, and should not act as a red herring to the clinician.

RADIOGRAPHY.

Radiography is an essential part of the examination, first in the diagnosis of the presence of an abscess, and secondly in its localization. While the plain radiographs may help clinch the diagnosis, fluoroscopic examination of the patient is the ideal. Only with the latter can that important feature in the diagnosis, limitation of movement or immobility of the diaphragm, be disclosed. Even the seriously ill patient can be examined under the fluoroscopic screen, for there is in the healthy person sufficient diaphragmatic movement in the supine position or in 10° to 15° of the Trendelenburg position to allow changes in mobility to be discerned. Should the patient be comparatively fit, he may be examined in the erect position.

Radiographs should be taken not only with the patient in the postero-anterior and lateral positions, but also in the lateral recumbent position, to facilitate localization. The films should be both soft and penetrating, the former to display any pulmonary changes and the latter to outline the diaphragm, which may be difficult to distinguish in the inflammatory mass of tissue.

Radiographic examination was carried out in 75 cases in the present series; 43 of these patients were examined fluoroscopically. However, such investigation is not infallible, and in three of the 75 cases in which abscesses were found at operation no abnormality was seen.

Radiographic findings may or may not be conclusive. Many changes seen are suggestive of the presence of an abscess; but the only truly diagnostic finding is the presence of gas and/or a fluid level beneath the diaphragm. Radiological interpretation may be divided into the following three sections: (*a*) pulmonary findings, (*b*) diaphragmatic findings, (*c*) subphrenic findings.

Pulmonary Findings.

There may be no alteration in the intrathoracic pattern, but often definite changes ensue, as occurred in 30 cases in this series. These are most commonly basal, and are usually reported radiologically by a variety of terms such as pleural reaction, pleural effusion, basal congestion, patchy areas of consolidation, and basal atelectasis.

Pleural Reaction: Pleural Effusion.

Pleural reaction is a sympathetic reaction to a neighbouring infection. There is initially a vague basal haziness obliterating the superior diaphragmatic surface and extending into the costo-phrenic sulcus, but lung tissue can be seen through it (Figure V). As it progresses, the resultant effusion obliterates completely the basal segments

of the pleural cavity. Occasionally it may fill most of the hemithorax. Of the 30 patients in this series with intrathoracic changes an effusion was proven on 11 occasions and suspected in a further six instances.

Basal Congestion: Patchy Areas of Consolidation: Basal Atelectasis.

Basal changes vary according to the severity of the underlying process. Initially the changes are associated with basal hyperæmia, but as basal compression increases and aeration diminishes, patchy consolidation appears. This may even progress further to bronchial obstruction, pneumonitis, and finally atelectasis. These last two processes were found on 17 occasions in this series. Harley (1949) describes as a common finding a triangular area of pulmonary collapse extending upwards and backwards in the paravertebral gutter towards the hilum, and seen best in the lateral view.

Comment.

These radiological findings in the thorax are evidently of importance. They should immediately suggest a nearby inflammatory process. On the other hand, they may be looked upon as primary and draw the clinician's attention away from the subphrenic region; or if they persist, or are interfered with, they may form the nidus for further complications, such as empyema.

Diaphragmatic Findings.

Characteristically the diaphragm is elevated and has lost its mobility. Normally the right side is higher than the left, so that elevation of this side is clearly noticed; but the left side, whose concavity is occupied by yielding soft structures, rises to less extent, and as it does so the clear distinction between the levels on both sides is lost (Figure V). Neuhof and Schlossmann (1942) pointed out that left-sided paresis occurs before elevation and should be regarded with suspicion. Diaphragmatic excursion may be considerably reduced or absent altogether. This may be due to the following factors: (i) direct paralysis of the diaphragmatic musculature by the toxic material which bathes its under surface, (ii) pressure effect due to the walled-off abscess in a confined suprahepatic space, (iii) reflex immobility to avoid spread of infection (Harley, 1949).

Unless the film is a penetrating one, the diaphragm may not be visualized. In health it is thin and well defined with a regular curve, but the presence of an underlying abscess leads to thickening, poor definition and tenting.

Thickening is due to the inflammatory changes in the muscle and the granulation tissue of the pyogenic membrane of the abscess laid down on its surface.

Poor definition is illustrated in Figure VI; the diaphragm loses its clear outline.

Tenting is shown in Figure VII; the diaphragm loses its regular contour and it may be more elevated or tented in one particular area. This is of considerable importance. Tenting suggests the site of greatest pressure, and therefore the site of the abscess, and should be of great help in deciding whether to approach the abscess from the front or the back.

Subphrenic Findings.

The only diagnostic subphrenic finding is the presence of gas and/or a fluid level (Figure VI), but it is a late finding (Table III). It was apparent in 24 cases (32%), so that in two-thirds of the cases there is nothing of significance; but if it is present, besides being diagnostic, it may aid in localization. The third radiograph, taken with the patient in the lateral recumbent position, will show the lateral and downward shift of the gas shadow and fluid level, and gives some idea of the extent of the abscess (Figure VIII). This picture is also of use should the patient be so ill as not to be able to sit up. If he is rolled on his side, a view can be taken with minimal disturbance to him. In this regard, Howkins (1948) points out that with the patient lying on one side the diaphragm pivots about the spine, the upper hemidiaphragm moving but little, the lower moving considerably.

Differential Diagnosis.

The differential diagnosis from other subphrenic gas shadows and fluid levels may be difficult in the presence of an elevated diaphragm. The most important of these are as follows.

Air under the diaphragm may follow a perforated viscus or persist for two or three weeks after a laparotomy. Usually the shadow is bilateral and the diaphragm thin and well defined (Figure IX).

The stomach bubble may be confused with a left-sided abscess. However, often the bubble will be displaced by the latter, usually medially. Further elucidation of the problem may be gained by fluoroscopic screening with the administration of a barium meal. This will demonstrate the presence of the gas shadow, distinct from the barium in the stomach.

TABLE III.

Incidence of Gas in Cases of Subphrenic Abscess.

Authority.	Number of Cases.	Percentage in which Gas was Present.
Ochsner and deBakey ..	25	30
Harley	135	27
Present series	75	32

Diaphragmatic hernia must always be considered in left-sided lesions, especially in the obese person and in one with changes in the lung base, in whom it is difficult to see the diaphragmatic level. X-ray examination with a barium meal and, if possible, fluoroscopic examination of the patient in the reverse Trendelenburg position will demonstrate the hernia. Such a differential diagnosis did not arise in this series.

Gaseous distension of the bowel, such as a volvulus of the pelvic colon, may cause elevation of the diaphragm by pressure and give the impression of gas under the diaphragm; but if a dry film is examined carefully, the colonic haustrations should be clearly visible.

A large perinephric abscess, especially on the left side, may lead to difficulty. However, careful clinical examination should produce the correct conclusions. A perinephric abscess, if large enough, may track in the extraperitoneal tissue plane and present the features of a subphrenic abscess. However, Neuhof and Schlossmann (1942) found an elevated paralysed diaphragm in only one out of 65 cases of perinephric abscess.

An intrahepatic abscess may be impossible to distinguish clinically or radiologically from subphrenic abscess. In this country a suppurating hydatid cyst must always be considered. In such cases the inflammatory reaction throughout the liver causes such hepatic enlargement that it is easy to obtain clinical evidence of enlargement downwards as well as upwards.

Transposition of viscera is a very rare cause of right-sided gas. The diagnosis should be clear if dextrocardia is noted, and X-ray examination with a barium meal will produce definite evidence of it (Pendergass, 1929).

Artificial Pneumoperitoneum.

Sante (1940) makes a plea for the use of artificial pneumoperitoneum as an aid in the radiological exploration of a subphrenic space. He has used it many times without spread of the infection or other injury to the patient. Only a small quantity of air is used, and the patient is radiographically examined in the erect or the lateral recumbent position. If the subphrenic space is clear, the under surface of the diaphragm becomes clearly outlined. If there is an inflammatory process beneath the diaphragm, this space will not be evident. This use of artificial pneumoperitoneum was helpful on one occasion

in this series, and positive results were obtained (Figure IV). Possibly artificial pneumoperitoneum has a small part to play, especially in those cases in which radiological examination fails to disclose subphrenic lesions as a basis for conditions found in the pleural cavity. Harley (1949) considered that it should not be used except in chronic abscesses, when the danger of rupture is much less, owing to thickness of the wall of the abscess and to the density of surrounding adhesions.

ASPIRATION OF CHEST OR SUBPHRENIC REGION.

Aspiration of the chest or subphrenic region has a very small part to play in either the diagnosis or treatment of subdiaphragmatic collections. Thorek (1947) considered aspiration at any time to be dangerous. Ochsner and deBakey (1938) and Harley (1949) stated that it should not be employed. Faxon (1940) considered aspiration of pus from a suspected area beneath the diaphragm to be a pernicious habit that should be condemned. There are several reasons for these statements, which may be set out as follows.

1. If pus is obtained through the diaphragm, infection of a previously clear pleural cavity is assured. The end result may not be empyema, for the infection may be controlled early by the body's resistance; but nevertheless there is a needle puncture in the diaphragm, and if the abscess is under pressure some pus must escape into the pleural cavity. Barnard (1908) describes a patient who died three hours after unsuccessful transpleural aspiration of a subphrenic collection of pus. The pleural cavity contained one and a half pints of pus, which had leaked through the diaphragmatic needle puncture. Nather and Ochsner (1923) suggest aspiration of a posterior abscess without involvement of the pleural cavity, by inserting the needle in the posterior axillary line at the level of the spinous process of the first lumbar vertebra and directing it upwards and backwards at an angle of not greater than 45°. Should pus be obtained the diagnosis is merely verified, and drainage has still to be carried out. In the present series 24 patients were subjected to transpleural aspiration, and fluid was obtained on 19 occasions. Pus was obtained on 11 occasions; in three of these latter cases, pus was already present in the pleural cavity, owing to rupture of the diaphragm in two cases, and to empyema caused by transpleural drainage in the third. Of the remaining eight patients, four subsequently developed empyemata, but in all cases this followed later transpleural drainage.

2. If serous fluid or nothing is obtained by aspiration, the diagnosis is no further advanced, and exploration has still to be carried out. Such was the state of affairs in 13 of the 24 cases in which aspiration was performed.

It can be clearly seen, therefore, that transpleural aspiration has little to offer as a safe procedure or reliable criterion in the diagnosis of subphrenic abscess.

PLEURAL EFFUSION.

As has already been shown, pleural effusion is a common occurrence with subphrenic infection. It is a sympathetic effusion, similar to that occurring in joints with periarticular inflammation. It was suspected radiologically or proven by aspiration or operation in 17 cases in the present series. The fluid is straw-coloured, and is sterile. It was aspirated on eight occasions in the present series, and of the five specimens examined by culture all were sterile. In general it amounts to five or six ounces, but occasionally it is produced in abundant quantities. Uncommonly the effusion may be bilateral, as occurred twice, or even contralateral, as on one occasion.

PLEURAL ASPIRATION.

Pleural aspiration is occasionally indicated if there is gross effusion with resultant dyspnœa. This occurred on two occasions in this series, and repeated aspiration of large quantities of straw-coloured sterile fluid, up to 28 to 39 ounces at a time, was carried out with great respiratory relief. It was only after subphrenic drainage in these two cases that the effusion slowly subsided. Care should be taken during the aspiration that the tip of the needle does not pass beyond the confines of the pleural cavity. Bailey (1948) sums up the whole question of aspiration very clearly, as follows:

> Needling for a subphrenic abscess should be forbidden absolutely. It may be quite justifiable to pass a needle into the pleural cavity to ascertain whether fluid is present and the nature of that fluid, but to pass the needle onwards in the endeavour to locate pus beneath the diaphragm is the quintescence of impropriety.

COMPLICATIONS OF SUBPHRENIC ABSCESS.

The complications of subphrenic abscess are of three varieties—thoracic, abdominal and metastatic.

Thoracic Complications.

Thoracic complications are by far the most common complications of subphrenic abscess, and account for much of the high mortality of the condition. As a general rule they arise through failure in diagnosis, or through meddlesome attempts to obtain one, but their incidence has dropped considerably since the universal employment of extraserous drainage of subdiaphragmatic collections. Several of the complications have been dealt with elsewhere, and only the more important of the remainder will be referred to here. Their incidence is set out in Table IV.

TABLE IV.
Thoracic Complications.

Complication.	Number of Cases.
Pleurisy	13
Pleural effusion ..	11
Pneumonitis	10
Atelectasis	7
Pneumothorax ..	4
Empyema	18
Lung abscess	6

Pneumothorax.

Pneumothorax occurred on four occasions in this series, and one patient died. In all cases it followed the opening of a pleural cavity. In only one of the four cases was infection not present. In one it followed transpleural drainage with adherent pleural surfaces, but this did not prevent the patient from developing pyopneumothorax. On two of these occasions the pyopneumothorax followed transpleural aspiration of the subphrenic collection.

The value of these figures is to impress upon the clinician the danger of entering a clean serous cavity, and the absolute necessity of extraserous drainage on all occasions when the infective process is limited to the under surface of the diaphragm.

Empyema.

There were 18 cases of empyema, and seven of the patients died. In nine of these cases the blame for the empyema could be laid at the door of the surgeon; it was the direct result of aspiration followed by transpleural subphrenic drainage. In two cases the cause was perforation of the diaphragm, with an early fatal result.

It is interesting to note that in this series there were no cases of subphrenic infection secondary to *empyema thoracis*. Harley (1949) records, out of 182 cases, only two in which empyema followed thoracic infection. Ochsner and deBakey (1938) estimated the figure at 2·5%.

Lung Abscess.

Lung abscess occurred in six patients, four of whom died. In two cases it followed transpleural drainage, and in the remainder it was due to hæmatogenous spread from the subphrenic region. In all cases it resulted in prolonged chronic illness, and the mortality rate was high.

Pericardial Effusion.

Pericardial effusion is a rare complication of subphrenic abscess. It did not occur in this series. Ochsner and deBakey (1938) put the incidence at 5%, but this is not borne out by other authorities. Spiers (1951), reviewing a case, considered it to be a sympathetic effusion, the rarity of which is due to the relation of the pericardium to the avascular central tendon of the diaphragm.

Abdominal Complications.

Apart from residual abscess in various regions of the abdomen associated in all four cases with rupture of the appendix, the abdominal complications resulted from local spread of the abscess.

On one occasion a liver abscess resulted. It is probable that this abscess was due to direct spread through the hepatic capsule from the abscess cavity, for it was in direct continuity with the subphrenic region and was drained through the same incision. The extension of a subphrenic abscess into the liver is very difficult to detect, because it is relatively uncommon. However, it should always be kept in mind if gross hepatic enlargement and jaundice are present, and if the patient's condition fails to improve after adequate drainage.

In one case there was diffuse toxic necrosis of the liver, due to the overwhelming toxæmia associated with a missed subphrenic abscess.

The third and most serious of the abdominal complications is intraperitoneal rupture of the abscess, which occurred on two occasions, both with fatal issue.

Metastatic Complications.

Metastatic complications occurred on three occasions, and took the form of pyogenic infection in the brain and kidney; all cases terminated fatally. In this series the subphrenic abscess passed through its surrounding wall on four occasions, in all cases with fatal results.

Such a complication must always be borne in mind when the question of operative or conservative treatment of an established abscess is raised.

Bacteriological Findings.

A culture was grown on 37 occasions. The streptococcus, the staphylococcus and the coliform organisms were roughly equally responsible for the infections. On several other occasions the attempted culture was unsuccessful, probably owing to the prolonged use of the chemotherapeutic and antibiotic agents.

Treatment of Subphrenic Abscess.

There is no doubt that the incidence of subphrenic abscess has lessened considerably since the advent of chemotherapy and the adoption of a more rational basis of surgical approach. Despite chemotherapy, in the established case surgical intervention is always indicated; but if the process has not progressed to suppuration, conservative measures should be adopted.

A subphrenic abscess as a localized entity takes a considerable time to form, and often the diagnosis takes even longer. Here again the antibiotics have often masked its presence, and posed even greater diagnostic problems. Chemotherapy may even cause many suspected subphrenic collections to abort, and the patient's routine convalescence may be only slightly longer than usual.

Initially, the patient is vaguely ill with irregular pyrexia and indefinite physical or radiological signs. Such a patient should be treated conservatively. Should these characteristic clinical manifestations of subphrenic infection continue with unabated persistence for periods up to a week, suppuration is probably present, and surgical drainage should be instituted without delay. Faxon (1940) goes so far as to state that the occasional unsuccessful exploration, early in the period when the subphrenic abscess is suspected, is far more desirable than procrastination carried to a degree that renders the presence and location of the abscess self-evident.

However, should symptoms and signs or radiological evidence be definite, and especially if pain is increasing or the patient's condition deteriorating rapidly, there should be no delay. Increasing pain indicates increasing tension within the abscess cavity, with subsequent danger of rupture and a steep rise in the mortality rate.

In this series, 75 patients were treated by drainage; 25 had no drainage. Of the former 13 died, and of the latter 20 died. Clearly, the higher mortality of the latter group indicates the gravity of procrastination, and the need for early and adequate drainage once the presence of an abscess is confirmed or strongly suspected.

Drainage.

The surgical approach depends on an accurate clinical and radiological assessment of the patient. A mass present

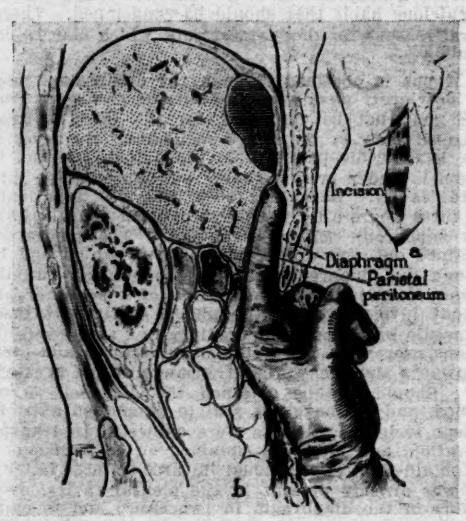

FIGURE X.

Illustration showing the method of extraperitoneally draining an abscess in the right suprahepatic space. As shown in (a), an incision is made below and paralleling the right costal margin, passing through the flat abdominal muscles and the transversalis fascia. By means of the finger the parietal peritoneum is peeled from the under surface of the diaphragm until the abscess cavity is reached. The abscess is then drained extraperitoneally without contaminating either the pleural or the peritoneal cavity. This also demonstrates the right subhepatic space with its postero-superior cul-de-sac. (From Ochsner and Graves, 1933, by permission.)

anteriorly suggests an anterior approach; œdema and redness posteriorly suggest a posterior approach. But all physical signs should be verified by radiographic examination with postero-anterior and lateral views, and all findings should then be correlated so that the correct approach is taken. In some cases doubt will still exist, and if one approach fails there should be no hesitation in carrying out exploration by another route immediately. The ideal type of drainage procedure should be one characterized by directness, simplicity, dependence, and avoidance of unnecessary contamination of uninvolved areas. There are two types of drainage procedures in use at the present time—transserous and extraserous. Both may be either pleural or peritoneal.

The transserous route is to be avoided if this is at all possible. There is no excuse for transgressing a virgin pleural or peritoneal cavity *en route* to pus. However, such a route may be the best, should an intrathoracic suppurative process already be present. But in general, it should be an absolute desideratum to institute that type of drainage which completely avoids the slightest chance

of contamination of these two virgin surfaces. Therefore, because of this possibility, the transpleural extraserous route should also be avoided. When this method was used, various attempts were made to obliterate the pleural cavity, artificially by suture, by irritating substances, or by mobilization of the costo-phrenic angle. But in many instances such precautions were inadequate in protecting the virgin pleural cavity against invasion. Harley (1949) records one death among four patients on whom drainage was performed through a pleural cavity which was stated to have been obliterated by adhesions. Barnard (1908) records five cases in which pleural adhesions gave way when the diaphragm descended, after the abscess had been drained. In this series a pyopneumothorax followed trans-

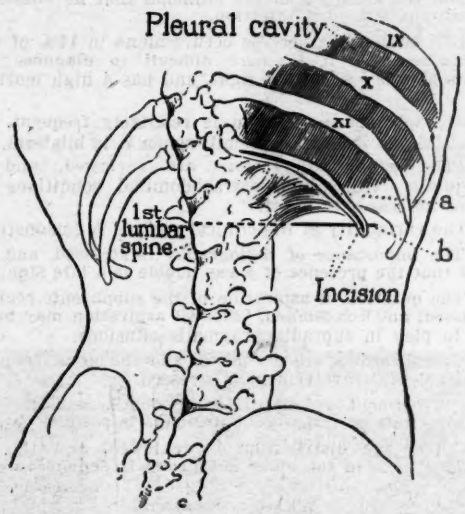

FIGURE XI.

Diagrammatic drawing showing skin incision made over and parallel to the twelfth rib, and transverse incision through the lumbar fascia and diaphragm at the level of the first lumbar spinous process. (From Ochsner and Graves, 1933, by permission.)

pleural drainage in a patient in whom the pleura was adherent. The route of choice, then, is an extraperitoneal one, and, according to the localization of the abscess, the approach may be anterior or posterior.

Anterior Approach.

The anterior approach was first described by Clairmont and Meyer (1926). It is a muscle-cutting incision just below and parallel to the costal margin. If the free edge of the liver and parietal peritoneum are not already adherent, as they often are, the latter is stripped off the diaphragm until the abscess is entered, through the adhesions walling it off from the peritoneal cavity (Figure X).

Posterior Approach.

Nather and Ochsner (1923) first demonstrated the posterior approach, and the ease with which posterior abscesses can be drained dependently without peritoneal contamination, a wound surrounded entirely by soft parts being left. These authors termed this a "retroperitoneal operation". The incision is made over and down to the twelfth rib (Figure XI), which is resected subperiosteally (Figure XII). At the level of the spinous process of the first lumbar vertebra, the musculature below the bed of the rib is incised transversely (Figure XIII), and in this way the pleura will be avoided, for it never comes below that level. Although the level of the costo-phrenic pleural reflection varies considerably in relation to the twelfth rib, it never extends below the level of the spinous process of the first lumbar vertebra, so that a transverse incision here is quite safe (Melnikoff, 1923).

The renal fascia, a smooth, shining, fibrous layer, can be seen readily, and through it the renal fat. From the point of view of operation, the peritoneum on the under surface of the diaphragm is continuous with the renal fascia, and they can both be stripped off in one layer. If an abscess is present, the peritoneum is very œdematous and the abscess easily opened. The incision needs to be large enough to admit a hand, because only in that way can one be certain that all loculi are broken down, and also to allow visualization and easy access to any intraperitoneal, perinephric or intrapleural collections. Of course, the danger of retroperitoneal operation is the accidental opening of a serous membrane, and failure to recognize such a complication.

These theoretical advantages of extraserous over transserous drainage are clearly substantiated in this series. Forty-six patients had extraserous drainage, of whom six died; 20 had transserous drainage, with a fatal issue on five occasions—this in the small series is nearly double the mortality of the extraserous method. In a much larger series Ochsner and deBakey (1938) record a 35·8% mortality rate in 721 cases of transserous drainage, but only 20·8% mortality in 211 cases of extraserous drainage. Harley's figures (1949) are 33% and 11% respectively, in a series of 125 patients treated by the two methods.

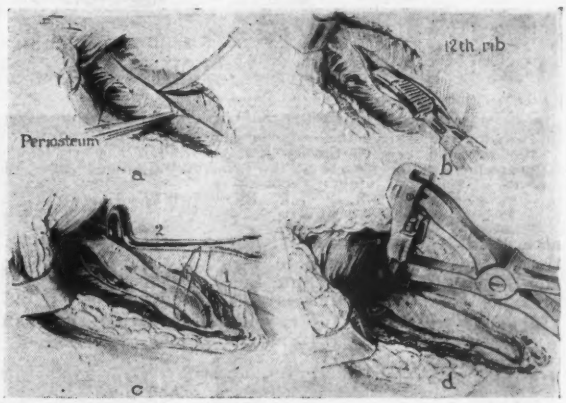

FIGURE XII.

Subperiosteal resection of the twelfth rib throughout its entire length. (From Ochsner and Graves, 1933, by permission.)

It is to be noted that there is a much higher proportion of cases in the present series in which drainage was carried out by the extraserous route, which is in contrast to those of other authorities. It is interesting to observe the lowering of the mortality rate over the last sixteen years, probably owing to increased cognizance of the condition, earlier diagnosis, and the use of the various antibiotic agents. It also appears that the overall extraserous drainage mortality rate will be difficult to lower below 11% to 12%, although the figures of individual surgeons will often be less, as their experience and operative ability increase. It can be seen clearly, then, that extraserous drainage is the method most consistent with good results and lowered mortality.

The route of the extraserous approach also appears to be important. Barnard in 1908 made the statement—and this was supported by Harley in 1949—that drainage was more safely performed from behind than from the front. It is probable that the close proximity of the general peritoneal cavity in anterior drainage accounts for this mortality. In this series the anterior approach was associated with twice the mortality rate of the posterior route.

After adequate drainage there should be an immediate improvement in the patient's condition. The gas and the fluid level may disappear within a few days, but the diaphragm may take much longer to recover, and

pulmonary changes may persist for months. In two cases in which clinical recovery occurred, X-ray examination two months after operation revealed the diaphragm to be elevated and still thick, and there was obliteration of the costo-phrenic angle.

If there is no improvement, or if improvement is followed by a regression, one should think of a further subphrenic abscess or loculus, or pulmonary sepsis, and treat it accordingly. In this regard it may be useful in the estimation of residual abscesses to inject a radio-opaque substance such as "Lipiodol" into the sinuses persisting from previous operative drainage. The ramifications of the "Lipiodol" will reveal the extent of the cavity, and may produce some surprises. In one case in this series the medium flowed from an abdominal sinus into a previously unsuspected empyema cavity.

FIGURE XIII.

Drawing illustrating the transverse incision made through the resected bed of the twelfth rib and the *serratus posterior inferior* muscle at the level of the spinous process of the first lumbar vertebra. (From Ochsner and Graves, 1933, by permission.)

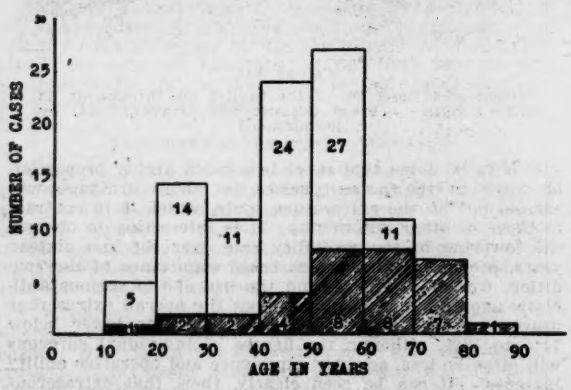

FIGURE XIV.

AGE DISTRIBUTION AND MORTALITY.

Reference to Figure XIV gives an interesting conception of the age distribution and overall mortality rate amongst the 100 cases reviewed. The forty to sixty years age group accounts for over half of the abscesses. Thirty-three of the cases had a fatal termination. Of these 33 patients, 24 were in the fifty to ninety years age group, a 52·2% mortality rate. The remaining nine were in the ten to fifty years group, a 16·7% mortality rate. This discrepancy in the mortality rate for these two age periods speaks for itself.

Clearly, the resistance of the older person to subphrenic abscess following a neoplastic process or operative trauma falls, and his chance of survival diminishes, as his age increases. In this series all eight patients aged over seventy years died.

SUMMARY.

1. The anatomy of the subphrenic region is reviewed, and only one suprahepatic space on the right side is recognised.

2. Right subphrenic abscess occurs commonly, and may so claim the attention of the clinician that he misses the concomitant left-sided infection.

3. Left subphrenic abscess occurs alone in 14% of cases in this series. It is more difficult to diagnose than subphrenic abscess on the right, and has a high mortality rate.

4. Multiple space infection is relatively frequent. Its mortality rate is high, especially when it is bilateral.

5. The ætiological factors are reviewed, and the infrequency with which extraabdominal conditions give rise to abscesses is noted.

6. The variability of the clinical picture is demonstrated.

7. The importance of radiography is stressed, and it is noted that the presence of a gas bubble is a late sign.

8. The question of aspiration of the subphrenic region is discussed, and condemned. Pleural aspiration may have a part to play in supradiaphragmatic effusions.

9. Complications are discussed, and the great frequency of intrapleural involvement is stressed.

10. Treatment is discussed in detail, and the high mortality rate of transserous drainage is pointed out.

11. The age distribution is reviewed, and the high mortality rate in the older age group is demonstrated.

ACKNOWLEDGEMENTS.

I wish to thank the Sister Superior of the Mater Misericordiæ Hospitals, and the Medical Superintendent of the Brisbane General Hospital, Dr. A. Pye, for permission to make use of the hospital records. I am grateful to Miss J. Clark for her photography, to Miss L. Tegus for her drawing, and to Miss M. Quigley for her invaluable assistance.

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ILLUSTRATION TO THE ARTICLE BY E. S. R. HUGHES AND R. A. JOSKE.

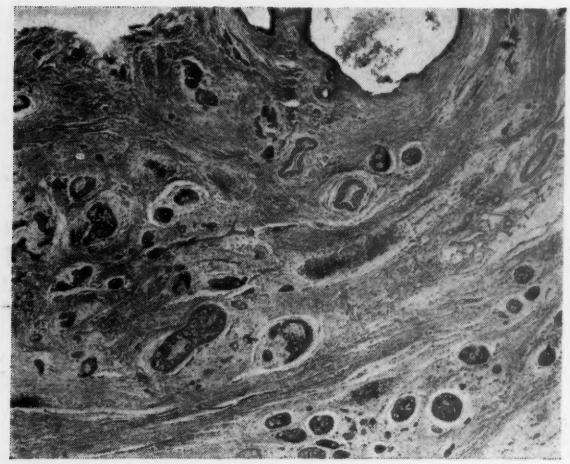
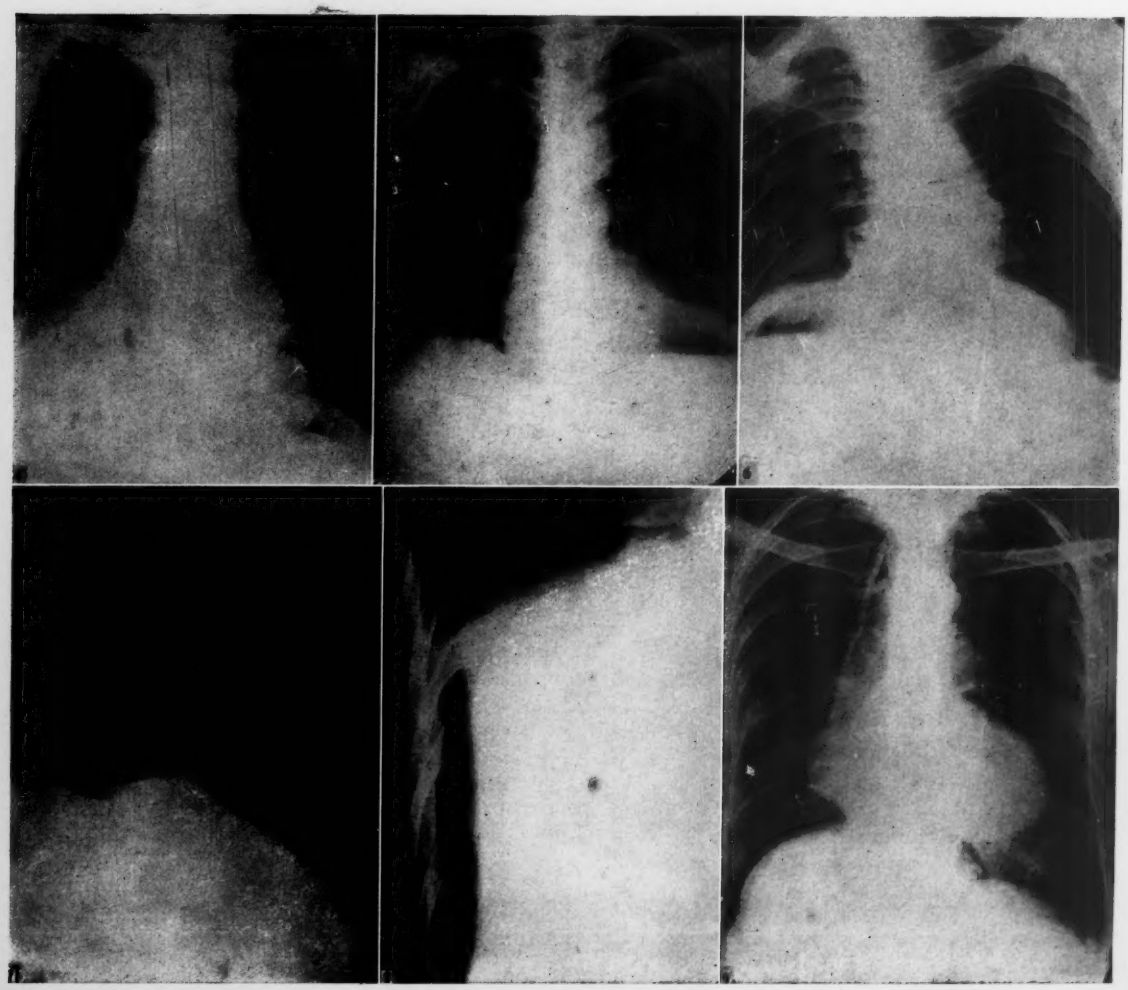


FIGURE III.

ILLUSTRATIONS TO THE ARTICLE BY C. J. WINDSOR, F.R.C.S.



ILLUSTRATIONS TO THE ARTICLE BY CYRIL FORTUNE, GORDON DONNAN, JOHN COLEBATCH AND THIES LUBBE.

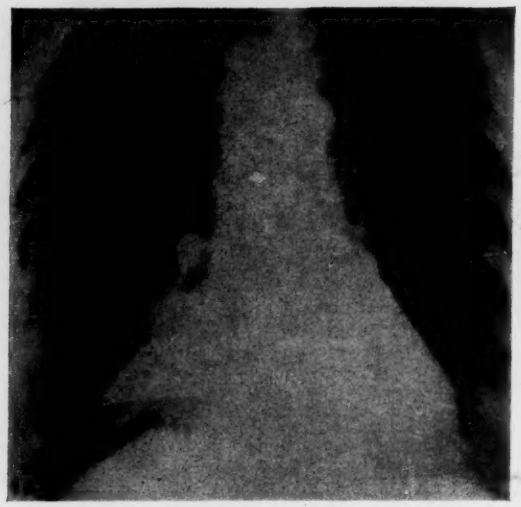


FIGURE IA.
Case I: Postero-anterior radiograph of an opaque area in the middle lobe of the right lung.

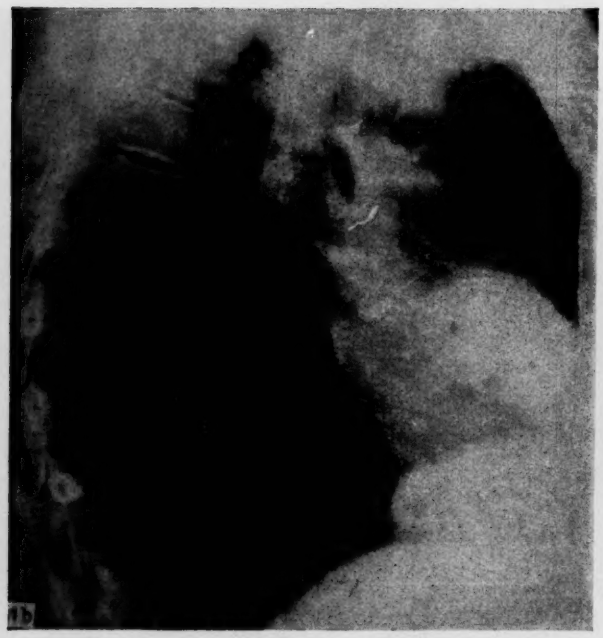


FIGURE IB.
Case I: Right lateral radiograph of an opaque area in the middle lobe of the right lung.

FIGURE III.
Case I: Photomicrograph of tumour shown in Figure II; a cryptococcus is conspicuous in the centre of this field.

ILLUSTRATIONS TO THE ARTICLE BY CYRIL FORTUNE, GORDON DONNAN, JOHN COLEBATCH AND THIES LUBBE.

FIGURE IV.
Case II: Radiograph of left knee, showing sclerosis of the proximal end of the tibia.

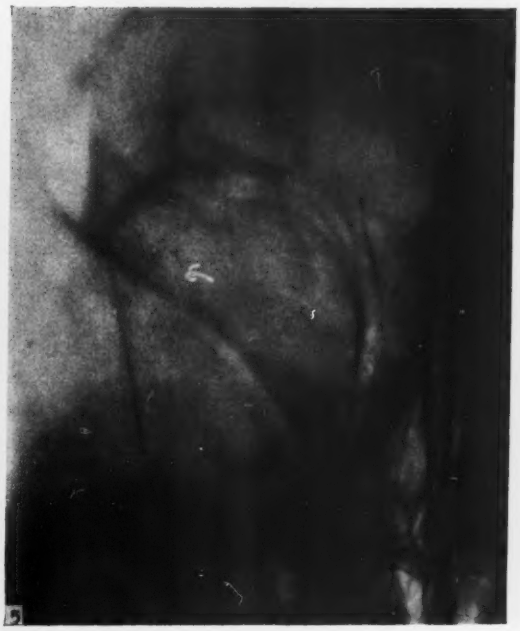


FIGURE V.
Case II: Radiograph of right orbit showing opaque area within an orbital tumour.

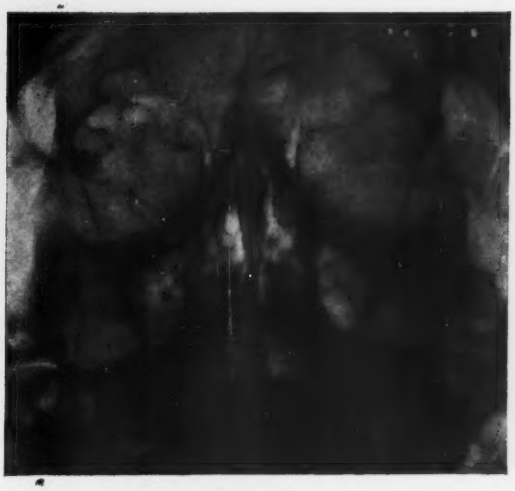


FIGURE VI.
Case II: Radiograph taken six months after that shown in Figure V, when opaque areas were present in both orbits.

ILLUSTRATIONS TO THE ARTICLE BY CYRIL FORTUNE, GORDON DONNAN, JOHN COLEBATCH AND THIES LUBBE.

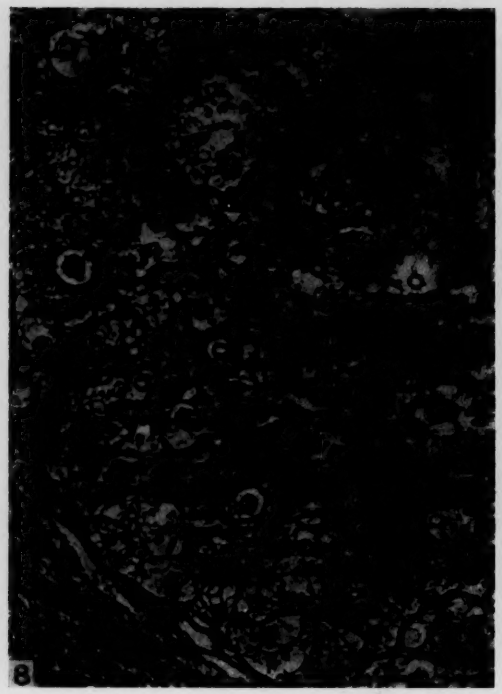


FIGURE VIII.

Case II: Photomicrograph of cerebral granuloma; many cryptococci are visible.

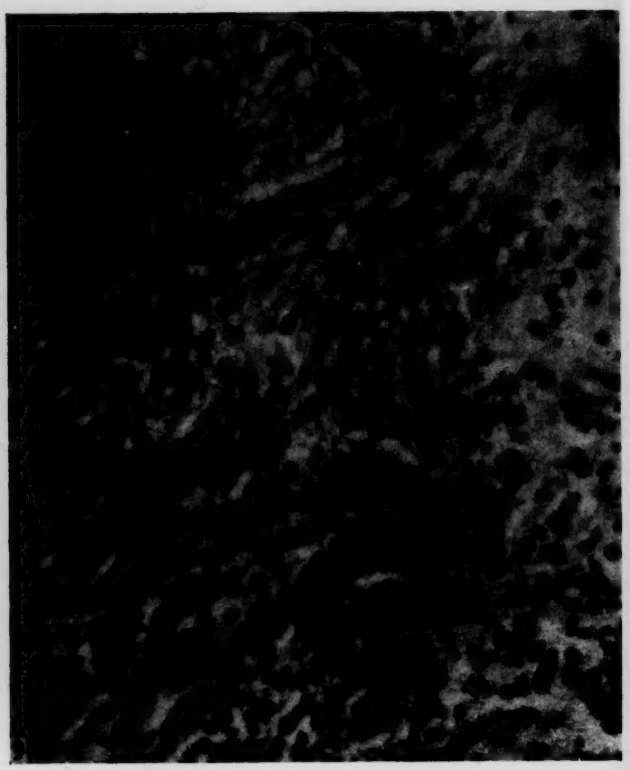


FIGURE IX.

Case II: Photomicrograph of section of cervical lymph node; many plasma cells and lymphocytes, no cryptococci visible.

ILLUSTRATIONS TO THE ARTICLE BY RICHARD HODGKINSON, M.S., F.R.A.C.S

FIGURE I.—Showing X-ray appearance on September 30, 1954, antero-posterior view. FIGURE II.—Showing X-ray appearance on February 15, 1954, antero-posterior view. FIGURE III.—Showing X-ray appearance on February 15, 1954, lateral view.

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Legends to Illustrations.

FIGURE IV.—Radiograph demonstrating loss of diaphragmatic outline and basal lung haziness. On the left side the left half of the diaphragm has been clearly outlined by an artificial pneumoperitoneum, but the right side is not evident.

FIGURE V.—Radiograph showing left subphrenic abscess. Note the relative equalization in the heights of the hemidiaphragms.

FIGURE VI.—Radiograph showing right subphrenic abscess with basal lung changes, elevation, thickening, and loss of definition of the diaphragm, and gas and fluid levels below the diaphragm. Note the difference in the heights of the diaphragm on the two sides.

FIGURE VII.—Radiograph demonstrating localized tenting of the diaphragm posteriorly at the site of greatest pressure. Note the fine pleural extension upwards. The two opacities are artefacts.

FIGURE VIII.—Radiograph demonstrating the extent and lateral shift of a subphrenic collection with the patient in the lateral recumbent position.

FIGURE IX.—Radiograph showing gas under the diaphragm on both sides. There are no pulmonary changes, and the diaphragm is thin, clear and regular.

Reports of Cases.

TORULOSIS.

By CYRIL FORTUNE, GORDON DONNAN, JOHN COLEBATCH AND THIES LUBBE,
Perth.

THE human disease torulosis is caused by the organism *Torula histolytica* (*Cryptococcus neoformans*). Related species of torulæ are widespread in nature, and pathogenic and non-pathogenic strains of *T. histolytica* have been isolated from the skin and intestinal tract of normal persons (Cox and Tolhurst, 1946). The cells of the pathogenic torulæ measure 1·0μ to 15μ in diameter. Budding, their only means of reproduction, is invariably evident. The buds have thinner walls than the mother cells and may be highly refractible. Surrounding the organisms in the tissues there is practically always a clear zone of gelatinous material. This constitutes a characteristic feature of the lesion. In artificial culture the morphological characteristics are practically the same, except that no capsules are formed.

The portal of entry of the cryptococci is obscure. Most observers consider it to be the lung. Wade and Stevenson (1941) considered there was a septicæmic stage. They were unable to produce central nervous system involvement without visceral involvement (chiefly lungs and kidneys) when they inoculated white mice intracerebrally, intravenously, intraperitoneally, intratracheally and subcutaneously. Intranasal inoculation produced severe rhinitis and sinusitis. The organisms spread out beneath the cribriform plate, but did not invade the central nervous system.

The suggested portal of entry is important in cases in which the only obvious site of involvement is the lungs, inasmuch as there is no specific therapy, and since pulmonary involvement may precede dissemination, surgical removal should be considered (Berk and Gerstl, 1952).

Histologically the miliary nodule, which resembles a tubercle both grossly and microscopically, is the commonest pathological lesion. Where there is a paucity of organisms the lesion may be a fibrotic granuloma. Where there is a massive proliferation of cryptococci, the lesion may be a gelatinous mass.

In regard to one of the cases to be described, it is of interest that Cox and Tolhurst have described calcified torulæ within the tissues of their experimental animals, but not in their human material. Sanfilice in 1895 described calcified torulæ in the tissues of an ox and also calcification of the tissues themselves, while Cox and Tolhurst have seen calcification in the tissues, once only, in the peritoneal node of a rat.

In 1894–1895 the remarkable Busse-Buschke case was described. The patient was a woman, aged thirty-one years, who for many years had had considerable enlargement of the lymphatic glands, and who had developed a large "abscess" of the tibia containing dirty brownish thick fluid. From this, yeast-like organisms were cultivated. At autopsy widespread lesions, often "purulent", were found in the pleural cavities, lungs, spleen, kidneys, bone, skin and left axilla. This is presumed to be a case of torulosis.

The first human case correctly diagnosed as due to torula infection was that of Verse (1914), who found in a woman, aged twenty-nine years, widespread leptomeningitis of the brain and spinal cord.

Stoddard and Cutler in 1916, by their careful studies, placed the pathology and clinical picture of torulosis on a firm basis. They gave the organism the name of *Torula histolytica*.

To date some 250 cases of torulosis have been described in the literature. Cox and Tolhurst in 1946 described 12 cases, and could find recorded a total of 35 cases in the Australian literature to that date.

CASE I.

Mrs. A., aged forty-seven years, presented on Januay 5, 1951, complaining of pain like a red-hot coal in the cervical part of the spine present for five weeks, passing to the scalp and into both temporal regions. Walking on uneven ground caused pain in the back passing round to the stomach. There was a complaint of loss of energy, anorexia, sleeplessness, tremor and giddiness. For three months she had coughed up blood-streaked phlegm in the early morning. Her chest had been radiologically examined six months previously, when she had a pleural friction rub in the right lower quadrant of the chest. This examination revealed an opaque area in the right lower lung field, which was considered to be due to a pleural effusion. Sputum tests at this time gave negative results for the tubercle bacillus.

On examination of the patient, the tonsils seemed to be very large, and pus could be expressed from the right tonsil. No significant abnormality was found on clinical examination of the respiratory and cardio-vascular systems. Pronounced neck stiffness was present. The *tendo Achillis* reflex was exaggerated, and ankle clonus was present. The plantar reflex was flexor in type. No glandular enlargement was seen. Radiographic examination of the spine and chest (Figures IA and IB) on January 9 revealed the following findings. Apart from lipping of the margins of the apophyseal joints between the fifth and sixth cervical vertebræ, the cervical and cervico-dorsal regions of the spine appeared normal. The lumbo-sacral part of the spine was of normal radiographic appearance for a patient of this age group.

X-ray examination of the chest gave the following findings. A postero-anterior projection showed a dense, homogenous roughly quadrilateral opaque area in the right lower lung field, continuous with the shadow of the mid-line structures. There was no evidence in this projection of disturbance of the normal vascular pattern to suggest collapse, nor was obvious obstructive emphysema present. Lateral projections of the chest and sternum demonstrated an ovoid opaque area extending from the anterior costophrenic recess into the lung field for a distance of about

eight centimetres; it appeared to be faintly reticulated, but it was thought that that might have been due to superimposed lung shadow. There was a suggestion that it extended below the diaphragm; no connexion with the hilum could be identified. There was no displacement of the interlobar fissures, nor was there definite hilar gland enlargement.

On the findings the following possible diagnoses were considered: (i) a benign neoplasm; (ii) a hernia of abdominal contents through a foramen of Morgagni or the intrathoracic extension of an hepatic abscess. A provisional diagnosis of spondylitis, chronic tonsillitis, carcinoma or pleuro-pericardial cyst of the base of the right lung was made, and on January 31 Dr. E. J. Clark performed a right middle lobe lobectomy.

Macroscopic examination of the resected lobe gave the following results. Near the hilum there was an oval tumour, approximately four by three centimetres in area, with a thin grey capsule. Its base fitted into the triangle formed by branching bronchi. The bulk of the tumour was

FIGURE II.

Case I: Macroscopic appearance of the middle lobe of the right lung, showing tumour near hilum.

composed of a soft, jelly-like, grey, opaque mass arranged in an alveolar pattern of irregular design. At the periphery were several small cavities surrounded by dense, grey, opaque tissue. A bronchial adenoma was diagnosed on this macroscopic appearance.

The photomicrograph (Figure III), taken from a section stained according to Crossman's method, showed several very conspicuous centrally situated unicellular organisms with clear capsules. Surrounding these capsules were polymorphonuclear cells, the exudate was hæmorrhagic, and alveolar septa were largely destroyed. The organisms were identified as *Cryptococcus neoformans*.

Examination of the cerebro-spinal fluid at this time gave negative results. In the post-operative period the patient complained of severe vomiting and constipation.

On March 22 the patient was still vomiting, her temperature was 101° F., and she was complaining of pain in the back of the head and neck. Examination of the cerebro-spinal fluid on March 25 gave the following results: the fluid contained 1100 leucocytes per cubic millimetre, 60% being lymphocytes and 40% polymorphonuclear cells; no organisms were detected, and an attempt at culture produced no growth after three days.

A further examination of the cerebro-spinal fluid on March 28 showed it to contain 270 leucocytes per cubic millimetre, 50% being polymorphonuclear cells and 50% lymphocytes; torulæ were present; an attempt at culture produced no growth after twenty-four hours.

The patient then returned to her home in the remote outback and died shortly afterwards.

Discussion.

Berk and Gerstl (1952) described a case of torulosis with solitary lung involvement, in which the patient was alive and well four years after lobectomy. In a review of more than 200 cases, they found only five other patients similarly affected. Our case does not qualify for addition to this small number, because before operation there were already symptoms (pronounced neck stiffness and alteration in the reflexes) suggesting neurological involvement. It was only to be expected that death would follow within a few months. However, this case is interesting in that the lesion simulated a tumour of the lung, which eventually led to lobectomy.

CASE II.

Mrs. B., a housewife, aged thirty-five years, presented in May, 1948, with a swelling behind the right upper eyelid and ptosis of the right eye. Her health had been good except for periodic attacks of pain in the left leg. These attacks first appeared in 1946, and the pain commenced just below the medial aspect of the left knee joint and extended down to the left ankle. A tender area on the upper medial aspect of the left tibia appeared to swell during attacks. The patient felt that the pain was in the bone, and between attacks there was no disability.

The right orbital tumour was removed in May, 1948, and in a report on a section of the excised tissue it was stated that "chronic inflammation with some lachrymal gland was present".

The patient remained well, but by November, 1948, proptosis of the right eye and a small nodule in the outer portion of the right eyelid were present. Some soft-tissue swelling was present on the medial aspect of the head of the left tibia. At this stage X-ray examination of the left tibia was reported as showing no abnormality, and X-ray examination of the orbit revealed no bony erosion; but an opaque area (discussed below) could be seen in the left orbit (Figure VI).

In February, 1949, the recurrent right orbital tumour was removed. Examination of a section of this was said to show "chronic inflammation with evidence of fibrosis".

In July, 1949, there was further recurrence of the right orbital tumour, with some proptosis and papillœdema; because of this the patient was referred to the Royal Melbourne Hospital in September, 1949, for aid in diagnosis and for neuro-surgical advice.

Dr. R. S. Hooper removed a well lobulated process involving some of the muscle of the orbit. Dr. E. King reported on the specimen that fibrous stroma was present with some spheroidal cells in groups lying in lymphatics; some large cells with large nuclei were present. He considered the condition to be an inflammatory pseudotumour of the orbit, as described in Dandy's monograph on orbital tumour, "Chronic Inflammatory Nodule". There was residual weakness of the right *levator palpebræ*, the superior rectus and the lateral rectus muscles from this operation.

The right orbital tumour recurred yet again and was excised in March, 1950. The pathological report on this tissue was as follows: "A chronic inflammatory lesion with many histiocytes and occasionally plasma cells, sometimes arranged around blood vessels: there is much fibrosis."

By April, 1950, there was an obvious swelling in the left orbit in the position of the lachrymal gland, similar to the tumour of the right orbit. There were recurrent attacks of pain in the left tibia. In view of dental sepsis, some teeth were removed in June, 1950, but this made no improvement in the patient's general condition.

In March, 1951, there were further symptoms (swelling and heat) suggestive of an inflammatory lesion at the proximal end of the left tibia. A course of streptomycin, 0·5 gramme twice a day for ten days, was without effect. At this stage the left lachrymal gland had ceased to enlarge and the right orbital tissue showed no evidence of recurrence of tumour; but the left leg remained warm over the upper medial aspect of the tibia.

Some investigations had been carried out during the previous three years and were repeated; there was no significant change over this period. The Wassermann and Kahn tests produced negative results. The Mantoux test produced a positive result with a dilution of one in 1000. The blood uric acid content was 6·0 milligrammes per 100 millilitres and the blood cholesterol content was 155 milligrammes per 100 millilitres. An X-ray examination of the chest showed the lung fields to be clear. An attempt at culture from the blood was unsuccessful. The hæmoglobin value was 10·5 grammes per 100 millilitres. The erythrocytes numbered 3,690,000 per cubic millimetre and were normocytic; the leucocytes numbered 12,900 per cubic millimetre, 84% being neutrophile cells, 7% monocytes and 9% lymphocytes. The erythrocyte sedimentation rate was 70 millimetres in one hour.

X-ray studies of the left tibia showed gradual changes over the three years. The tibia was reported as normal on two occasions prior to March, 1951, when increased density with some irregular areas of partial translucency were noticed in the head of this bone, suggestive of an old inflammatory lesion.

In April, 1951, the appearances were those of an irregular amorphous sclerosis of the proximal end of the tibia, most pronounced postero-laterally (Figure IV). Plain films and tomography failed to demonstrate bone destruction; the bone was not expanded or deformed. The evidence was consistent with low-grade osteitis, and *Brucella abortus* infection was suggested. Serological studies gave some support to this suggestion (see Table I).

TABLE I.

Tests for Brucella Abortus Agglutination.

Date (1951).	Titre.		
	1 in 100.	1 in 200.	1 in 400.
April 24 ..	+	∓	—
April 30 ..	+	−	Trace.
July 21 ..	+	+	Trace.
August 13 ..	+	±	Trace.

In May, 1951, a lymph gland anterior to the right parotid gland was enlarged to one inch in diameter. It was excised. Microscopic examination revealed infiltration with histiocytes, plasma cells and occasional lymphocytes, much fibrosis, and some giant cells with a single nucleus, sometimes with several nuclei. A fungous infection was considered and further sections were stained in an effort to demonstrate fungi, but none was found.

In the following few weeks the glands of the upper right cervical chain enlarged as discrete lumps; a thyroid swelling was palpable; lymph glands in the inguinal region enlarged, and small glands in both axillæ were palpable. At times the tip of the spleen could be felt. The left leg was warm and swollen, just below the knee, and the patient was pyrexial.

In June, 1951, a course of "Aureomycin" was given, two grammes daily for seven days, then one gramme daily for fourteen days. There was no benefit from this. After this, at the end of June, a course of deep X-ray therapy was tried on the glands in the right posterior triangle of the neck, again with no effect and no benefit to the patient.

Thus, by August, 1951, when the patient had been observed for three years and three months, the diagnosis was as obscure as ever. On the whole the evidence favoured a chronic inflammatory rather than a neoplastic disease. The pathologist was very close to the correct diagnosis when he considered a fungous infection, but even so he was unable to confirm this idea in May, 1951. Only for *B. abortus* infection was there any confirmatory evidence, and in view of the sustained titre of one in 200 for this organism, it was decided to give intensive treatment with "Chloromycetin" as outlined by Woodward *et alii* (1949). The course given was six grammes daily for seven days, followed by three grammes daily for four weeks.

Just as the course was proposed, the patient became ill with a severe pain in the centre of the chest and a temperature up to 103° F. The next day an obvious pericardial friction rub was heard from the apex to the base of the heart. Within four days of the commencement of intensive "Chloromycetin" therapy the temperature subsided, and on the eighth day an X-ray film of the chest showed a normal picture for heart and lungs. In retrospect, it is considered that the pericarditis may have been caused by the torula organisms, and "Chloromycetin" may have been significant in the rapid defervescence and the transient nature of the friction rub.

With the foregoing treatment there was continued improvement and relief of discomfort in the left leg; but the patient became very nauseated, and diarrhœa began on the day when the course was completed. There was no decrease in the size of the glands. During this period the hæmoglobin value fell to 8·5 grammes per 100 millilitres; two pints of blood were given.

When the patient was examined in October, 1951, the left orbital tumour had enlarged, there was no recurrence of the right orbital tumour, the condition of all other glands remained unaltered, and she was apyrexial. Agglutination for *B. abortus* was reduced to only ± for a one in 50 dilution.

She was admitted to hospital in January, 1952, for removal of the left orbital tumour, which had continued to enlarge and prevented any upward movement of the globe. On examination of the patient, there was no general change in her condition. Treatment included the administration of ACTH, 10 milligrammes four times a day for twenty days to a total of 800 milligrammes, and "Aureomycin" (no "Chloromycetin" was available), three grammes daily for twenty days to a total of 60 grammes.

The left orbital tumour was removed by Dr. J. P. Ainslie on February 14. Diarrhœa and occasional vomiting were considered to be due to "Aureomycin" and lasted for about one month. Three weeks after her admission to hospital the patient complained of an aching pain below the left knee, which disappeared in five days with only symptomatic treatment. Studies of the erythrocyte sedimentation rate showed a gradual fall from 119 to four millimetres in one hour whilst she was receiving cortisone, with a subsequent rise to 125 millimetres in one hour after this therapy ceased. Serial white cell counts showed no significant change or abnormality, except that values remained persistently high (10,000 to 17,000 per cubic millimetre). Examination of sections of the tumour revealed a picture essentially similar to those previously reported; plasma cells were noted as predominant and often aligned along capillaries, and fibrous tissue was invading striated muscle.

About this time diagnostic aid was sought from experts overseas, and sections with relevant information were sent to them. Professor Dorothy Russell considered that there were points inconsistent with a diagnosis of multiple myelomatosis, Sjorgen's disease, or Schüller-Christian's disease, and favoured a diagnosis of subacute progressive pyogenic infection with a reaction of hyperimmunity evidenced by plasma cell infiltration. Both Dr. George Lumb and Dr. Norman Ashton agreed in regarding the picture as one of chronic inflammatory reaction of unknown ætiology. All suggested that a biopsy and attempted culture of the material from the left tibia might be helpful. However, this latter investigation was not carried out because at this time symptoms referred to the left tibia and local changes were only mild, and X-ray changes in this area were not such as to suggest that biopsy would be profitable.

X-ray examination of the left knee and tibia in February, 1952, showed no change from the previous year. Tomographs did not show a cavity in the left tibia.

The patient was discharged from hospital, and readmitted in May, 1952, with a history of gradually increasing swelling of the left thigh and leg. There was generalized lymphadenopathy; for the most part firm, mobile, discrete and non-tender glands were present; masses of glands were especially prominent in the submandibular region

beneath the right sternomastoid and in the left inguinal region.

Her last admission to hospital was on February 3, 1953, when she gave a history of a severe frontal headache present for the previous four days. For six months both nostrils had been blocked, the left more so than the right. Enlarged lymph glands were still present; there had been little change in eight months. The left leg remained slightly swollen and warmer than the right over the upper end of the tibia.

A blood examination gave the following results. The hæmoglobin value was 11·5 grammes per 100 millilitres; the leucocytes numbered 14,500 per cubic millimetre, 93% being neutrophile cells, 3% band forms, 1% monocytes and 3% lymphocytes. Repeated estimations showed no significant variation in these values during the next three weeks.

Lumbar puncture on February 4 produced clear, colourless cerebro-spinal fluid under a pressure of 170 millimetres of fluid, with no block; the protein content was 59 milligrammes per 100 millilitres, there was no excess globulin, and the amount of reducing substances was 59 milligrammes per 100 millilitres; the chloride content (as sodium chloride) was 709 milligrammes per 100 millilitres; cells numbered 46 per cubic millimetre, 45% being polymorphonuclear cells and 55% lymphocytes; the cerebro-spinal fluid was sterile. Lumbar puncture was repeated on February 14 and produced slightly turbid cerebro-spinal fluid under a pressure of 400 millimetres of fluid; the protein content was 66 milligrammes per 100 millilitres; the amount of reducing substances was 39 milligrammes per 100 millilitres, and the chloride content was 689 milligrammes per 100 millilitres; cells numbered 419 per cubic millimetre, 60% being polymorphonuclear leucocytes and 40% lymphocytes; no organisms were detected in a smear of the cerebro-spinal fluid, and attempted culture yielded no growth of microorganisms. X-ray examination of the skull and sinuses (February 5) revealed almost complete opacity of the left antrum, with diminished translucency of the left frontal and ethmoidal sinuses. Further X-ray examinations on February 13 and 23 revealed increasingly opaque sinuses, but no abnormality of the bone. An X-ray examination of the chest on February 21 showed that the left hilar shadow was more prominent than the right; the lung fields were clear. The heart shadow was reduced in size compared with previous films. No Bence-Jones protein was present in the urine.

The plasma protein content was 7·6 grammes per 100 millilitres—albumin 2·1, globulin 5·5 grammes; pseudoglobin (α), 1.3; fractionation euglobulin $\beta.\gamma$, $\frac{2\cdot5 \quad 1\cdot7}{4\cdot2}$.

Headache was controlled with "Pethidine" and a course of nitrogen mustard therapy was commenced; in all 17·5 milligrammes were given in four days. Soon after this "Pethidine" was no longer required and the patient seemed very well. However, she was kept in hospital, and a fruitless effort was made to overcome the cause of her persistent pyrexia with "Chloromycetin" (two grammes daily). Soon afterwards her mental state deteriorated, and she became rapidly worse without any specific symptoms and died on February 26, 1953.

Post-Mortem Findings.

The autopsy was carried out by one of us (T.R.L.). The body was emaciated, with healed operation scars on the left side of the face (orbit and parotid region). There was a chain of slightly enlarged, firm lymph glands on the right side of the neck; examination of sections of these revealed a rather fibrous grey cut surface with ill-defined minute brownish grey areas. In the mucosa of the right side of the pharynx there were several small flat ulcers, a result of X-ray irradiation. In the lungs the only abnormalities detected were non-specific bronchopneumonic lesions. The right and left tibiæ contained fatty, fibrous bone marrow.

The most conspicuous abnormality was intracranial. The meninges of the base were turbid and gelatinous. The bases of both temporal lobes felt nodular; the nodules were only a few millimetres in diameter and could be felt better than seen. Both middle fossæ had a most peculiar moth-eaten appearance (Figure VII), due to small hemispherical cavities with bases facing the meninges and convexity pointing downwards into the bone; they were often arranged in small groups and irregularly scattered across both middle fossæ. These cavities were not seen in other parts of the skull, and seemed to correspond in size and position to the nodules felt in the brain. Their origin was puzzling, but they were most probably a result of the pressure of the nodules and pulsation of the brain.

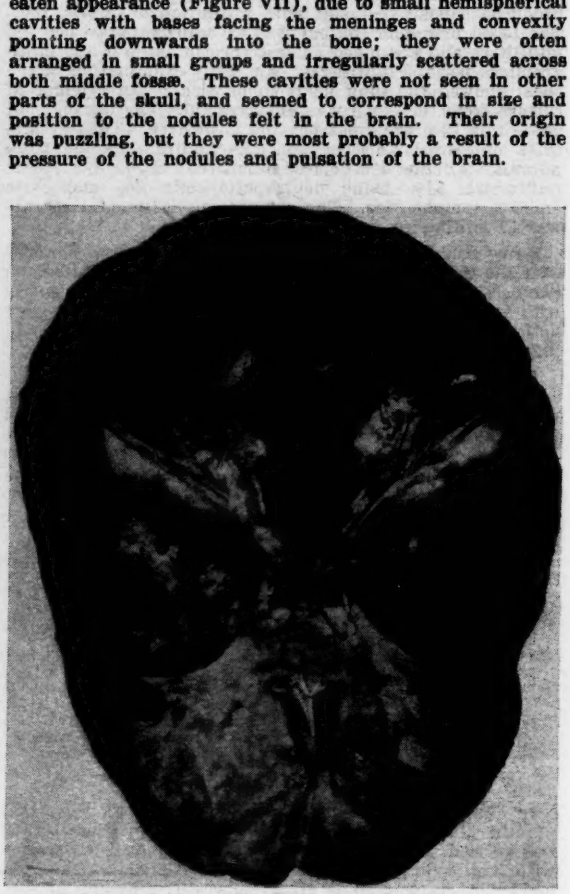

FIGURE VII.
Case II: Photograph showing bony erosion in both middle fossæ.

The cerebro-spinal fluid was turbid, and in a smear (Indian ink) several cryptococci were seen as large clear circles about 10μ in diameter, in the centre of which was a double-contoured organism of circular shape.

The ethmoidal cells were opened and contained a gelatinous mass, which had to a large extent destroyed the normal constituents of the cavity. No organisms were found in a smear from this mass.

No lesions were found in the pericardium or heart.

Histological Findings.

Meninges.—In the meninges a considerable amount of inflammatory infiltration was apparent; the cells in the exudate were mainly histiocytes and occasional plasma cells with scattered polymorphonuclear cells; lymphocytes were fairly numerous. In the neighbourhood of capillaries, fibroblasts were proliferating and often coalescing to form multinucleated giant cells with peripherally arranged pale vesicular nuclei; their nuclear membrance and faint chromatin network were apparently of different age from that of the giant cells found in the cervical lymph nodes. The cytoplasm of these giant cells was pale pink (hæmatoxylin and eosin stain) and very finely granular; occasionally a round organism about 7μ in diameter was found in the cytoplasm displacing one or two nuclei. The exudate

varied in character in different fields of vision; fibroblastic proliferation alternated with a more cellular, less organized exudate. The organisms were usually round, sometimes ovoid, with nearly always a double contoured and smooth outer rim and a pale or more often unstained capsule. Examination of sections stained with Crossman's stain revealed the organisms as brilliant light green disks surrounded by an unstained or lightly stained capsule 2μ to 3μ in diameter.

Brain.—Examination of a section of the brain revealed small granulomata which contained many organisms (Figure VIII). The surrounding reaction was insignificant. Here and there a few compound granular corpuscles were identified. Organisms in this section showed a few spikes radially arranged across the capsular zone, as if suspending the central organism.

Pituitary.—The pituitary gland was unaffected. However, its upper surface was surrounded by chronic granulation tissue containing many plasma cells and histiocytes in about equal proportions. There was considerable fibroblastic reaction, and a few relatively small multinucleated giant cells contained six to seven nuclei radially arranged surrounding a foamy and otherwise structureless cytoplasm.

Cervical Lymph Node.—Examination of a section of a cervical lymph node (Figure IX) revealed diffuse infiltration with chronic inflammatory cells in a wavy background of rather acellular collagen fibres. Most of the cells were lymphocytic, but again many plasma cells were present, and occasionally multinucleated foreign body giant cells. Cryptococci could not be found. In several further sections substantially the same picture was seen; the destruction of normal lymphatic architecture was very striking.

Pharynx.—No fungi were found in sections of the pharyngeal ulcers, which were probably the result of irradiation. Dense superficially necrotic connective tissue formed the ulcer base, beneath which was loose connective tissue containing chronic inflammatory cells. In the underlying straited muscle there was some fibrosis, with proliferation of sarcolemma nuclei.

Lungs.—The right lung and the upper lobe of the left lung were congested and œdematous. The lower lobe of the left lung was heavily consolidated, and examination of a section revealed non-specific bronchopneumonia, with many abscesses composed of polymorphonuclear cells and occasional foamy histiocytes. Much destruction of alveolar walls had taken place. No fungi were detected.

Adrenal Glands.—Nothing arousing suspicion of torulosis was seen in the adrenal glands. There was a small necrotic cortical infarct, a result of thrombosis of small vessels.

Bone Marrow.—In the left femoral bone marrow abnormal cellularity was present. There were many histiocytes and plasma cells. However, no fungi were found.

Tibial Bones.—Several specimens were taken from both tibiæ; none of them showed any evidence of torulosis. The lesion of the left tibia had been treated with X rays, and that may have been locally effective. Examination of sections showed only pronounced fibrosis of the bone marrow with no significant cells or configuration of cell groups.

Summary.

To summarize the macroscopic and microscopic findings: only in the meninges and brain were cryptococci detected. None could be found in the lungs. In the absence of positive proof, one can only surmise that the lesions found elsewhere and described above were caused by torulæ.

Discussion.

The diagnosis was not established until the post-mortem examination. In retrospect, it is difficult to see how the correct diagnosis could have been made, when efforts to demonstrate fungi in numerous sections were unsuccessful. At the post-mortem examination cryptococci could be demonstrated only in the brain, meninges and cerebrospinal fluid. It was suggested more than once during the long course of the patient's illness that biopsy of the abnormal area of the left tibia would have been helpful; but this is unlikely in view of the necropsy findings.

Overall, this patient with her periodic pyrexia, slight leucocytosis, granulomatous lesion of lymph glands and plasma cell infiltration of tissues presented a picture of chronic infection. Since this is one way in which torulosis may present, and probably a not uncommon way, it seems worth keeping such a diagnosis in mind when the cause of a chronic inflammatory reaction is difficult to diagnose.

Natural History and Treatment.

The long course that torulosis may take is well illustrated by this case. The patient was observed for five years, and if one assumes, as seems reasonable, that the tibial changes were another manifestation of cryptococcal infection, then the duration was at least seven years. This compares with the longest survival in reported cases—between seven and eight years (Voyles and Beck, Case IV, 1946; Holmes and Hawks, 1953). Death followed one month after symptoms of central nervous system involvement appeared. Many patients do not come under observation until neurological symptoms are present, and death usually follows within three or four months (Cox and Tolhurst, 1946). However, Marshall and Teed (1951) reported a case of torulosis with central nervous system involvement—proved by cerebro-spinal fluid culture—in which cure followed treatment with sulphadiazine and potassium iodide; this is the only recorded case in which torulosis of the central nervous system could be considered cured. Mosberg and Alvarez-DeChoudens (1951) from in-vitro experiments reported that alkalinization and hyperthermia might be of value. However, the patient treated by Stimson and Dean (1952) with sulphadiazine and alkali died seven weeks after the onset of symptoms. Sulphonamides and potassium iodide had no apparent effect in Symmers's (1953) Case I, and the organism was found to be insensitive to sulphonamides and antibiotics *in vitro*. Most clinical and experimental work suggests the inefficacy of potassium iodide, sulphonamides and antibiotics. "Aureomycin" and "Chloromycetin", with the possible exception of the latter in treatment of the pericarditis, were without effect in the treatment of our patient.

Ocular Aspects.

Henry L. Birge (1952), with a comprehensive review of the literature, discussed ocular aspects of mycotic infection. Though involvement of the eye is rare, the literature on such cases is extensive. In general, fungi produce a superficial lesion of the eye and surrounding skin in addition to possible systemic lesions. The conjunctiva is often affected and the naso-lachrymal duct obstructed. He found that Weiss in 1948 described the only known case of intraocular cryptococcosis. The first affected eye showed a "cystic growth" of the retina and was enucleated; three months later the same process destroyed the sight of the second eye. *Cryptococcus neoformans* was cultivated from fluid obtained when the sclera was trephined.

No case could be found of enlargement of lachrymal glands due to torulosis or any other fungous infection. We regard this as the first such case to be reported.

Relation to Hodgkin's Disease.

Not infrequently patients who are subsequently found to have torulosis are diagnosed as suffering from Hodgkin's disease, and the question arises whether these two diseases are coincident or causally related. Cox and Tolhurst, in a review of such published cases, found that in none was there a picture indubitably that of Hodgkin's disease, and in several the data given were inadequate for a firm diagnosis. Usually all the features could be accounted for by an infection with *Cryptococcus neoformans*. Hodgkin's disease was diagnosed erroneously in the cases of Symmers (1953, Case I) and of Holmes and Hawks (1953), before torulosis was discovered. This diagnostic confusion is not surprising, for Hodgkin's disease occupies a position intermediate between the infective granulomata and

malignant tumours, and in different cases the histology varies from the pleomorphic picture of granulomata to the more invasive and uniform cellular picture of a sarcoma. Lymph gland enlargement, which occurs in about 18% of patients with torulosis, may be diagnosed as due to Hodgin's disease, chiefly because no other condition can be thought of to account for the picture; but usually the histological features of the glands are not characteristic of Hodgkin's disease—there are no Dorothy Reed-Sternburg giant cells, but only foreign body type giant cells, and fibrosis is more pronounced. However, in chronic forms of torulosis in which lymphadenopathy may occur, sometimes a condition develops which is identical with Hodgkin's disease.

Cryptococci, it seems, induce hyperplasia of the reticulo-endothelial system in a fair proportion of cases, and as Cox and Tolhurst suggest, occasionally this may undergo malignant transformation into Hodgkin's disease or lymphatic leuchæmia. Thus these authors regard one of their own cases of torulosis (Case VII) as being a case of Hodgkin's disease as well, and concede that some form of lymphoblastoma has been associated with torulosis in about 5% of reported cases.

Even more obscure is the reported association of torulosis with myeloid leuchæmia. In the case of torulosis of Ring and Williams (1952), the patient presented with a typical picture of myeloid leuchæmia two years and ten months before the onset of neurological symptoms; and the authors quote Zelman *et alii* (1951), who found 14 cases in which the ante-mortem diagnosis of chronic myeloid leuchæmia was firmly established. Such an association could hardly be coincidental, but the causal relation is not clear.

At no stage in this patient's history was there evidence of involvement of the lungs; nor were cryptococci found in sections of the lungs taken at the post-mortem examination. This is unusual, since the lungs are considered to be the mode of entry in most cases.

Neurological Features.

Necropsy revealed a typical diffuse cryptococcal meningo-encephalitis. The meninges were gelatinous, and there were many cerebral granulomata. In the temporal lobes these granulomata had caused multiple small erosions of the bone of the middle fossa.

Radiological Findings.

Certain features were of particular interest, and one, calcification of gland masses, we believe has not previously been described.

Plain X-ray films of the tibia (November 22, 1948) were reported as showing no abnormality; but later review indicated possible incipient changes. These changes were progressive, until ultimately an irregular amorphous sclerosis of the upper end of the tibia, more pronounced postero-laterally with a little periosteal lift over the lateral aspect of the sclerosed area (Figure IV), was seen in April, 1951. Plain films and tomography failed to demonstrate bone destruction. The bone was not expanded or deformed. The appearances suggested a low grade osteitis, and the radiologist suggested a brucella infection.

Collins (1950), in a review of more than 200 cases of torulosis in the literature, found 17 with bone involvement. He reported three additional cases with X-ray observation over fifteen months. The characteristic radiological feature in these was a discrete osteolytic lesion, which was often disseminated.

Examination of the orbit on May 4, 1948, revealed at the site of swelling in the right orbit a somewhat amorphous deposit of calcium (Figure V). There was no evidence of bone erosion in the vicinity. The tumour was mainly radiolucent and did not appear to involve bone.

At examination on November 22, 1948, an opaque area of similar form and situation to that seen in the right orbit, but less dense, could be identified within the shadow of the left orbit (Figure VI). That these opacities of calcium density seen within the shadow of the orbit were within the tumour mass was confirmed by their disappearance on operative removal of the tumours.

Terminally, there was complete opacity of the left antrum, left ethmoid cells, left frontal sinus and left sphenoid sinus, in contradistinction to their complete radiolucency at previous examinations. Post-mortem examination showed this to be due to invasion by gelatinous granulation tissue.

Cryptococci were cultivated from the naso-pharynx of the patient of Rappaport and Kaplan, and at autopsy a purulent exudate containing cryptococci was found in the left sphenoid sinus.

At no stage of this patient's radiological investigation was there evidence of pulmonary involvement.

Summary.

Two cases of disseminated torulosis are described.

One patient presented with an opaque area in the middle lobe of the right lung, suggesting a tumour, which proved when resected to be a gelatinous mass containing typical cryptococci.

The second patient was observed for five years, during which period there was generalized glandular enlargement, including both lachrymal glands, and a sclerotic lesion of the left tibia. The X-ray changes in the left tibia differed from those which usually occur in cryptococcal bone lesions. Pericarditis was a brief though unusual feature of her illness, and agglutination for *Br. abortus* to a dilution of one in 200 was an odd finding during investigation. The duration of the disease was at least seven years.

Diagnosis was not established until after operation in the first case, and not until necropsy in the second. Both patients died soon after symptoms of central nervous system involvement became evident.

Acknowledgements.

We are indebted to Dr. F. Clark, who performed a lobectomy in the first case, and to Dr. J. P. Ainslie and Dr. R. S. Hooper, who excised lachrymal glands from the second patient. We are grateful for the assistance of Dr. John Day, who was responsible for the ophthalmic care of the second patient, and to Professor Ida Mann, who helped to further the diagnosis in this case.

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BRUCELLOSIS OF THE LUMBAR SPINE: REPORT OF A CASE.

By Richard Hodgkinson, M.S., F.R.A.C.S.,
Sydney.

This case is reported because no bone lesions diagnosed as being caused by *Brucella abortus* have been noted in the Australian literature.

Case History.

Mr. A., grazier, fifty-seven years of age, of western New South Wales, was first seen on July 27, 1953. He complained of back pain of one month's duration. He first noticed the pain after cranking a small stationary engine. The pain radiated from the small of his back into both groins and into his right buttock. It was aggravated by coughing and by train travelling. It was relieved by rest and moderate walking.

On examination his spine was found to be straight and his lumbar spine was flattened. All spinal movements were reduced, and forward flexion produced pain in the left buttock. Straight-leg raising of the left leg produced pain in the left buttock at 90°. Ankle and knee reflexes were normal and there was no sensory loss. Medial rotational movements of both hips were reduced and caused pain on the left side. Skiagrams showed degenerative spondylitis with narrowed intervertebral spaces between the first and second lumbar, fifth lumbar and first sacral vertebræ, and between the fourth and fifth cervical vertebræ. Numerous Schmorl's nodules were visible. He was fitted with a Taylor's brace.

His pain persisted and on September 30, 1953, it was noticed that his cremasteric reflexes were absent. A skiagram on this date, the tube being focused on the first and second lumbar area, showed irregular narrowing of the disk space and an irregularity of the upper anterior lip of the body of the second lumbar vertebra (Figure I). Blood counts, test for hydatid, and chest X-ray examination all gave normal results.

In January the patient's back pain was still troublesome. Skiagrams of the area in his spine showed increase in the development of the destructive lesion in the second lumbar body and the formation of large antero-lateral osteophytes bridging the disk space to join the first lumbar body to the second (Figures II and III). In February he was admitted to hospital and further investigations showed no abnormality, except that *B. abortus* was agglutinated in a titre of 1 in 32 and 1 in 64. This test was repeated by the Department of Health, which reported that agglutination occurred against *B. abortus* up to a dilution of 1 in 640.

The patient then volunteered the information that he had bought dairy cattle from the south of New South Wales which had suffered from "contagious abortion" in December, 1952, and also that he had treated a horse for "fistulous withers", a chronic suppuration beneath the *ligamentum nuchæ*, at the base of the neck. Veterinary surgeons inform me that this is usually caused by *B. abortus*. He had been treated in the country in January, 1952, for atypical pneumonia, which had lasted seven weeks; few details of this illness were available. In May, 1953, a few weeks before the first examination, he had had what was supposedly a recurrence of this illness and had had an irregular fever. He is at present being treated with two grammes of streptomycin daily and one gramme of aureomycin six-hourly; this latter dose was not well tolerated. He is free of pain.

Discussion.

The first case of undulant fever diagnosed in Australia occurred in a Maltese and was reported by Storie Dixson (1908). The first indigenous case in the State of New South Wales was reported by Tebbutt and Marsh (1931); this occurred at Kempsey, in a North Coast dairying district. Since that time veterinary officers have been active, but *B. abortus* is still common among cattle in New South Wales.

Bishop (1939) gives a very thorough review of the cases of bone involvement caused by this group of organisms, and reports the fifty-seventh case to that date. He notes that localization in the spine seems to be the commonest bone and joint complication. He points out that there has been no report on the pathology of this condition in man, but that in hogs the process seemed to originate in the epiphysis and formed irregular abscesses in the bodies and disks of the vertebræ, and that a crescent-shaped cap-like inflammatory proliferation of osteogenic tissue bridged the intervertebral space on the ventral surfaces of the affected vertebræ.

King-Brown (1952) again reviewed the literature and reported two further cases of brucellar spondylitis. In view of the prevalence of *B. abortus* in this country, and the recognized high incidence of bone involvement elsewhere, the possibility of brucellosis as a cause of backache should always be considered.

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Reviews.

Amputations. By Leon Gillis, M.B.E., M.B., B.Ch. (Witwatersrand), M.Ch. Orth. (Liverpool), D.L.O. (London), F.R.C.S. (England), F.R.C.S. (Edinburgh), Hon. F.I.C.S. (Geneva); 1954. London: William Heinemann (Medical Books), Limited. 10″ × 7½″, pp. 434, with 342 illustrations. Price: 84s.

This excellent volume fills a long-felt want as a book of reference and for post-graduate study. The arrangement of its subject matter is good; it is easy to read, whilst the illustrations are well conceived, faithfully reproduced and are most instructive. The book is composed of 20 chapters and these include one on "Amputations for Gangrene and Vascular Diseases" and 44 pages on congenital anomalies of the limbs and their management, apart from the many other facets covered most lucidly. "Painful stumps" are well discussed, as regards both ætiology and treatment, and the book concludes with chapters on the nursing of the amputee and anæsthesia for amputations. Orthopædic and traumatic surgeons should possess this volume if for no other reason than for its bibliography, which fills some 60 pages and covers every aspect of the subject.

Potassium Metabolism in Health and Disease. By Howard L. Holley, M.D., and Warner W. Carlson, Ph.D.; 1955. New York: Grune and Stratton. 9″ × 6″, pp. 144, with nine illustrations. Price: $4.50.

Although the importance of the potassium ion in body metabolism has been known for many years, it is only during the last few that the functions of potassium in the body have been studied in detail and the importance of deficiency and excess of potassium in the production of disease processes has been appreciated. In this small book of 115 pages in the "Modern Medical Monographs" series, H. L. Holley and W. W. Carlson have endeavoured to present what is known of the function of potassium in the body and the diagnosis and treatment of abnormalities of potassium metabolism. Quite a considerable amount of work has been done on the relation between potassium in the cells and the energy-yielding reactions. The importance of potassium ion in the functioning of the enzyme systems of the cells is shown in some detail and, on the whole, very clearly. It is obvious from these considerations that deficiency or excess of potassium within the cells must have important consequences on the health of the body. It is shown, too, that extracellular potassium plays a very important part in the regulation of the body functions. Although only 70 to 77 milliequivalents of potassium are found in the inter-

stitial fluid and blood plasma out of a total of 4000 to 5000 milliequivalents in the whole body, it is changes in the extracellular concentration of potassium which are of the greatest importance in the effects of potassium deficiency and excess. Sixty-two pages are given to consideration of the effects, diagnosis and treatment of abnormal potassium metabolism. Much use is made of accounts of illustrative cases with detailed comments. This part is very well done. Finally there is a useful appendix on "Units Used in Body Fluid Measurements" and a section gives sample low potassium and high potassium diets; this is followed by a very complete list giving the potassium content of foodstuffs.

This little book could be read with advantage by all physicians and is recommended for anyone who wishes to read a short but detailed account of potassium metabolism.

The Practice of Refraction. by Stewart Duke-Elder, K.C.V.O., M.A., D.Sc., Ph.D., LL.D., M.D., D.M., F.R.C.S., F.A.C.S.; Sixth Edition; 1954. London: J. and A. Churchill, Limited. 8" × 5½", pp. 348, with 239 illustrations. Price: 21s.

THIS book remains essentially the same as previous editions with the expansion and inclusion as a special chapter of the section on contact lenses. This is full of good advice and includes a small section on the recent innovation, corneal lenses.

Several illustrations have been changed for more modern ones and the author has changed the reading test type from the old J-system of Jaeger over to the more recent N-system recommended by the Faculty of Ophthalmologists.

The book is pleasing to read and is highly recommended to all refractionists.

The Digestive Tract in Roentgenology. By Jacob Buckstein, M.D.; Second Edition; 1953. Philadelphia: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. Volume I: "Introduction, the Hypopharynx and the Esophagus, the Stomach, the Duodenum." 10" × 7½", pp. 446, with 422 illustrations. Price: £13 8s. 9d. (two volumes).

THE second volume (which contains the index) of this work has already been reviewed in these pages. This volume, the first, deals with the hypopharynx, œsophagus, stomach and duodenum.

The author first considers the act of swallowing and describes its mechanism and the action of the various muscles. Incoordination of the muscle groups in aged women and in various anæmias is often the cause of dysphagia without organic disease. Webs in the pharynx are sometimes seen in these cases, which are often described as *globus hystericus*. Pharyngeal lesions, including pouches, are very well illustrated. In making œsophageal examinations, the author advises starvation for ten hours before the patient is seen, in order to obviate mistakes from retained food. A thick bolus is used and this, owing to its slower passage, generally allows good views to be obtained; this is followed by a liquid meal, which is better in cases of obstruction. The examination is made in various positions, but the author prefers the prone position for films.

Every known pathological condition is described and well illustrated, especially the variety of diverticula which have been met with in the author's practice. Cardiospasm receives much attention and is considered to be due to degenerative changes in the plexus of Auerbach. Several cases of webs near the lower end of the œsophagus have been reported. In foreign body cases, air may at times be seen when the foreign body has perforated the organ. Several cases of peptic ulcer of the lower part of the œsophagus are reported. These are due to regurgitation of acid gastric secretion.

In the author's experience, short œsophagus and thoracic stomach (not true hiatus hernia) are by no means uncommon. A preliminary fluoroscopic examination should precede the meal in gastric cases. A watery suspension of barium sulphate is recommended. Extragastric and intrinsic lesions are all described in minute detail and well illustrated. The author considers the differentiation between ulcer and carcinoma to be most difficult. He describes his methods in dealing with this problem. Many rare lesions are described and illustrated.

The author is not over-enthusiastic about the use of mass radiography of the population for early carcinoma. Only 1·24 lesions per thousand were discovered in a series of 2432 people who had no marked dyspeptic symptoms. Better results might be obtained in marked dyspeptics. In the author's experience the diagnosis of gastric syphilis and tuberculosis depends on the discovery of the organisms concerned. The section on the post-operative stomach is worthy of study. The differentiation between duodenitis and ulcer is difficult and attempts should always be made to show a crater or niche. The rarity of ulcer of the second part of the duodenum is noted, while ulcer is unknown in the third part. In the latter region any lesion found is carcinomatous. The book is most readable, as the author is a fluent writer and is able to describe clearly the cases met with in his enormous experience. The book is well worth close study and can be considered the best work on the subject yet received.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Medicine for Nurses", by M. Toohey, M.D., M.R.C.P., D.C.H., with a chapter on Psychological Medicine by Henry R. Rollin, M.D., D.P.M.; Second Edition; 1955. Edinburgh and London: E. and S. Livingstone, Limited. 8½" × 5½", pp. 656, with 180 illustrations. Price: 28s.

Intended to help the nurse during training and to be a reference book afterwards.

"Any Wife or Any Husband: A Book for Couples Who Have Met Sexual Difficulties and for Doctors", by Medica (Dr. Joan Graham); Second Edition; 1955. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 147. Price: 10s.

The title is self-explanatory.

"Textbook of Gynæcology", by J. H. Peel, M.A., B.M., B.Ch. (Oxon.), F.R.C.S., F.R.C.O.G.; Fourth Edition; 1955. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 506, with 206 illustrations. Price: 27s. 6d.

The text has been carefully revised.

"Materia Medica and Pharmacology for Nurses", by J. S. Peel, M.P.S., with a foreword by Flora Cameron, O.B.E., with Key to the Calculations, for the use of lecturers and tutor sisters only; 1955. Christchurch: N. M. Peryer, Limited. 8½" × 5½", pp. 176, with 14 illustrations. Price: 24s.

"The book attempts to present the nurse with a balanced picture of modern drug therapy without an undue amount of detail."

"A Molecular Conception of Organisms and Neoplasms: A Theory that any Organism is Basically a Single Chemical Molecule, Put Forward as the Key to the Problem of the Causation of Neoplasms", by T. L. Cleave, M.R.C.P. (London), with a foreword by C. P. Stewart, D.Sc. (Dunelm), Ph.D. (Edinburgh); Third Edition; 1955. Bristol: John Wright and Sons, Limited. 8½" × 5½", pp. 28, with two illustrations.

The title is self-explanatory.

"Spot Diagnosis: With Notes on Therapy", compiled by the editors of *Medicine Illustrated*; 1955. London: Harvey and Blythe, Limited. Volume II. 8½" × 5½", pp. 128, with 100 illustrations. Price: 8s. 6d.

This is "simply a small book attempting to stimulate and refresh the mind on diagnostic problems, in an entertaining manner".

"Doctor Against Witchdoctor", by E. W. Doell, with illustrations by Con Purchase; 1955. London: Christopher Johnson. 8½" × 5½", pp. 216, with many illustrations. Price: 15s.

Based on personal experience during many years of practice in African tribal areas.

"Aids to Surgical Anatomy", by D. B. Moffat, M.B., B.S., F.R.C.S. (England), and J. S. Baxter, M.D., M.Sc., F.R.C.S.I.; Fourth Edition; 1955. London: Baillière, Tindall and Cox. 6½" × 4", pp. 242, with 48 illustrations. Price: 8s. 6d.

One of the well-known Students' Aids Series.

"Research in General Practice", by James M. Mackintosh, M.D., LL.D., F.R.C.P. (Edinburgh and London); 1955. Edinburgh: T. and A. Constable, Limited. 8½" × 5½", pp. 58.

The First John Matheson Shaw Lecture delivered in 1954 at the Royal College of Physicians.

The Medical Journal of Australia

SATURDAY, AUGUST 6, 1955.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words of phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

CALCIUM INTAKE AND RICKETS.

ALTHOUGH standards of intake of calcium have been laid down and are in general use, the present position as to the daily requirements of calcium by children and adults is far from satisfactory. The signs of calcium deficiency are uncertain, and we do not know, although much is surmised, that a low calcium intake will lead to rickets. One table of recommended daily dietary allowances gives for calcium 0·8 gramme for women and men, 1·0 to 1·2 grammes for children from infancy to twelve years, and 1·4 grammes for children from thirteen to nineteen years with increased amounts for pregnancy and lactation. The figures are based on balance experiments done some years ago. The total number of such balance experiments considered in making the assessments was small, and the experiments were of short duration. The uncertainties of interpretation of balance experiments are well known, and an examination of the figures used shows a wide variation in apparent needs. Many factors can upset balances, especially in the cases of calcium, of which there is a large reservoir in the bones. At most the figure chosen is a preferred one and tells us nothing about the optimum intake or the lowest intake before symptoms appear. There is increasing evidence throughout the world that a great many people have a daily intake over periods of years which is much less than the supposed requirements. The dietary surveys carried out under the ægis of the Nutrition Committee of the National Health and Medical Research Council showed that many people, especially in the interior of Australia, had intakes very much less than the standard, and yet Australian country men are noted for big, solid bones. The Report to the Parliament of the Commonwealth of Australia, "Food Consumption Levels in Australia and the United Kingdom", drew attention in 1945 to the fact that the food available for civilian consumption in Australia in 1944 could not supply the amount of calcium necessary to fulfil the standard requirements. Subsequent reports from the Commonwealth Statistician show that the position has been very similar in the years that followed. In many regions of the tropics and semitropics the calcium intake has been shown by several observers to be invariably low as compared with recognized standards. A standard for intake is useful, provided one does not use it too rigidly.

A high intake of calcium is often recommended on the grounds that a low intake causes or predisposes to the development of rickets. In investigations carried out at the South African Institute of Medical Research, Johannesburg, A. R. P. Walker[1] has considered whether a low intake of calcium does cause or promote the development of rickets. One of the first difficulties to be considered is the diagnosis of rickets. Severe rickets is easy enough to diagnose, but there is little unanimity among clinicians about the recognition of the milder forms of rickets. Radiographical assessment of long bones has been much used, particularly by nutrition workers studying rickets or supposed rickets. It is recognized now that poorly mineralized bone can arise from many causes and does not necessarily indicate either rickets or inadequate calcium intake. Histopathological assessments of sections of the costo-chondral junction of a rib have been used in the diagnosis of mild rickets. Unfortunately the gradings used by different workers have varied so much that their results are not comparable, and some assessments are so severe that almost all the children in a community might be diagnosed as rhachitic. Chemical analysis of bone is almost as useless. In severe rickets there is a diminution in the amount of calcium and phosphorus in the bones, but Walker has shown that in the fifth rib of South African Bantu infants, children and adults the mineralization data, in cases of mild clinical rickets, lay within the same range as for non-rhachitic subjects. The deliberate attempt to induce rickets in infants is out of the question, and the results of experiments on lower animals cannot be transferred to human infants. The experimental assessment of the relation of low calcium intake to the incidence of rickets in infants cannot well be made. The effect of calcium supplements only on rickets is also very difficult to assess. The experiment must be carried on for some time, and it would be very difficult to maintain a constant exposure to radiation. We have, too, no knowledge, for example, of calcium stores in the body and of the effects of uncontrolled factors on the release of calcium from the stores or *vice versa*. That severe rickets can occur in the presence of ample calcium has long been known. Wake has shown in

[1] *Am. J. Clin. Nutrition*, March-April, 1955.

Sydney that infants who have had abundant calcium from birth, with long daily exposure to sunlight and in many cases extra vitamin *D*, may show radiological evidence which has been diagnosed as rickets. A study of calcium intake in relation to the incidence of rickets has been made by Walker. "It must be constantly kept in mind that rickets is a disease of *growing* bone, for among children whose growth is retarded or arrested, rickets is not observed. Now all diets which support growth *must* have a modicum of calcium. If calcium intake be decreased, what point is reached first? Is it the point where insufficiency of the element causes bone to be poorly mineralised, or is it the point where the concomitant lack of other nutrients (calories, proteins etc.) causes a slowing down of growth, thereby automatically arresting the development of rickets?" Wartime experience has shown that when all nutrients, including calcium, were severely restricted to the extent of starvation, rickets was not observed. Walker has examined the incidence of rickets in two classes of population. The first live in the tropics and semitropics, where the intake of calcium is habitually very low, but where available radiation is plentiful. The second group live in northerly countries, where calcium intake is higher, but where available radiation is not plentiful. Clinical rickets is rare in rural districts in East Africa, Rhodesia, Indonesia, Burma and India. In India and South Africa rickets is common in the crowded centres of population. The farm-working Chinese women, on a very poor diet, do not acquire osteomalacia; the well-to-do Chinese women living indoors on a better diet do. Walker concludes that there is here no evidence that a low calcium intake causes or promotes the development of rickets or osteomalacia. A number of studies in Northern Europe suggest indirectly that the level of calcium intake is of little relevance in the incidence of the disease as compared with low radiation. For example, in a study in wartime Denmark when food supplies were poor Fredericia reported that the incidence of rickets in the first year of life was 22% in the spring, but only 1·4% in the summer and autumn. Work in Glasgow by Findlay and Ferguson in 1919 gave similar results—although the diet consumed by the non-rhachitic children was only slightly superior to that of the rhachitic children; of the latter 40% were not taken out regularly, while only 4% of the non-rhachitic children had been confined indoors regularly.

The amount of calcium intake must have some effect on the incidence of rickets, but low calcium intake *per se* does not cause rickets. Vitamin *D* is said to save calcium, that is, the more vitamin *D* that is taken the less calcium is required. There must be a limit to this, of course, when there is not enough calcium available for absorption to supply the needs of the growing bones. Still, the actual calcium requirement is probably very low in comparison with the commonly used standards, provided enough vitamin *D* is obtained either from the food or better from radiation. There is certainly no evidence for a high calcium intake. The observations that have been published in several places in the last few years on the so-called idiopathic hypercalcæmia of infants suggest that high calcium intakes have their dangers.

Current Comment.

MICROBIAL DISEASES: UNSOLVED PROBLEMS.

WITH the great improvements in hygiene over the past century, the introduction of antitoxins and vaccines, the development of the "sulpha" drugs and the antibiotics and other drugs, infectious and other microbial diseases are in many parts of the world no longer the causes of vast numbers of deaths. It is not so long since Osler called pneumonia the captain of the men of death, and typhoid fever, cholera, plague and smallpox took toll of millions. Now there is a tendency to believe that infectious diseases have been conquered. However, René J. Dubos[1] voices a very timely warning that this is far from true and that there are many unsolved problems. If the number of deaths due to microbial diseases has been very greatly reduced, there still remains a vast amount of ill health, discomfort and loss of working time due to microbial infections.

> Mortality rates do not constitute adequate yardsticks to measure the importance of medical problems. If one were to use as criteria the amount of life spoiled by disease, instead of measuring only that destroyed by death; or the number of days lost from pleasure and work because of so-called minor ailments; or merely the sums paid for drugs, hospitals and doctor's bills, the toll exacted by microbial pathogens would seem very large indeed. Microbial diseases have not been conquered.

A great many manifestations of ill health are due to organisms which are usually not thought of in considering microbial diseases and are called non-pathogenic. What are usually considered the most typical bacterial poisons are the exotoxins of diphtheria and tetanus. There are, however, many constituents and products of micro-organisms which do not manifest themselves by dramatic effects but can cause slow, low-grade intoxication, and these play a more important part in the production of disease now than do the classical exotoxins. Although the ideas of Metchnikoff on intestinal intoxication have long been out of favour, there is probably a not inconsiderable amount of truth in them. Organisms from what is called the normal flora of the intestines from time to time pass in small numbers into the circulation and produce, usually, transient bacteriæmias, which are deleterious to health, although not lethal. The introduction into the circulation of small quantities of the cellular constituents of Gram-negative bacteria can be responsible for many disease conditions from minor febrile effects to disturbed sugar metabolism and pathological changes in the blood vessels. A rather striking indication of the action of apparently normal intestinal flora has been shown recently in the effects on the growth of young animals and even human babies produced by the oral administration of small amounts of antibiotics. The available evidence is strongly in favour of the view that the antibiotics alter the intestinal flora and thus prevent the absorption from the gut of deleterious bacterial products. In other words, retardation of growth, without any other obvious signs or symptoms, can occur in young animals on an adequate diet because of a mild and constant intestinal intoxication. Pathogens of all kinds—animal parasites, protozoa, fungi, bacteria, rickettsias and viruses—can persist in the tissues of a susceptible host without causing detectable disease. This can be of importance in connexion with disease in several different ways. These infections may remain latent for long periods producing no obvious symptoms. But some physiological or pathological change may occur, equilibrium is upset, and mild or even severe manifestations of the disease associated with the infective agent appear. The virus of *herpes simplex* gives good examples of this. Infections with this virus may occur at an early age and persist through life with few or no manifestations. But the equilibrium between host and virus is unstable, especially in certain parts of the body; and minor disturbances can upset it, and herpes blisters appear. This is

[1] *J.A.M.A.*, April 23, 1955.

a simple, relatively unimportant, example, but no doubt others, more serious, could be found if looked for. Tuberculosis infection in some cases fits this picture. The monilial infections following antibiotic treatments afford another example, as do the acute staphylococcal infections of the intestines after antibiotic treatment. Another important aspect of latent infections is the possibility of transferring organisms from the immune host to fresh persons who are not immune, with the production of overt disease. Most infectious diseases are caused by micro-organisms that are widely distributed among normal, apparently healthy members of the population. What is the nature of the immunity in the carrier of the latent infection we do not know. In the case of the intestinal organisms, such as monilia and staphylococci, it is probably not a bodily immunity at all, but the production by other intestinal organisms of substances which inhibit the unrestricted growth of the monilia, staphylococci *et cetera*. Why carriers are not always infectious is not known, but obviously the knowledge of this might have great importance in disease prevention. A great deal is still to be learned of the physiological factors of the host, in microbial infection, that are concerned in the development of lesions and symptoms.

Very little is known of the precise mechanisms by which microorganisms cause disease. It is known that the α-toxin of *Clostridium welchii* is a lecithinase, but how this acts in the body of an infected person is not known. The diphtheria toxin interferes with the synthesis or function of the cytochrome system, which is an essential in very many tissue oxidations. In the past toxic manifestations have been studied almost exclusively as immunological events, but this does not help us to understand toxæmia. A knowledge of the precise mode of action in the body cells of microbial toxins could be of great service in the study of improved methods of treatment of microbial diseases.

An aspect of infectious diseases which must have increasing importance in the future is related to the rapidity of travel nowadays. A person infected with some microorganism can travel great distances while still in the highly infectious stage and infect people thousands of miles from the original source of infection. This is seen well with new strains of influenza or poliomyelitis viruses; in the days of slower travel these might remain at the seat of origin long enough to have lost much of their virulence, but now they can travel rapidly to a distant community with no immunity. Here a tribute should be paid to the excellence of the quarantine service maintained by the Commonwealth Department of Health—the original health activity undertaken by the Commonwealth as such soon after its inauguration.

We may conclude by quoting *verbatim* the summary with which Dubos concludes his contribution.

> In summary, it seems to me that the center of interest in the field of infectious diseases is likely to shift during the coming decades. Now that many of the problems that haunted the medical and lay mind of the 19th century have been solved, it is necessary to formulate new questions more relevant to the times. Scientists believe that they understand the process of infection, but they are rarely able to prevent its occurrence. They publicise widely, and with justified pride, the very low death rates now exacted by the great killers of the past, but neglect to mention the enormous amount of disease caused by the microbial pathogens and also by micro-organisms that are not usually regarded as pathogenic.
>
> It is unlikely, I fear, that much progress will be made in the near future towards eliminating the micro-organisms that cause the diseases now endemic. Indeed, the fact that most members of the population will continue to harbor microbial agents that constitute a potential threat to health, even though not necessarily to life, must be accepted. Better techniques to prevent or at least correct the damage that they cause can certainly be developed. More important, the factors that permit so many individuals to harbor, without manifestations of disease, micro-organisms endowed with pathogenic potentialities can be eventually understood. On the basis of this knowledge it might some-day become possible to design procedures of metabolic control that will help man live at peace with the micro-organisms. These micro-organisms, after all, do constitute an inescapable part of his biological environment.

CHEMOTHERAPY OF CANCER.

THE search for drugs which will influence the growth of malignant tumours and related blood conditions has been very intensive over the past ten years, but no real cancer cure has yet been found. While it does not seem very likely that any drug will be found which will cure all types of malignant growths, because of the great variation in the structure, metabolism and ætiology of different growths, there has been some success in the alleviation of symptoms, slowing down of the rate of growth and even retrogression of tumour growths. F. Bergel[1] has considered the types of chemical substances which have shown some success, particularly those in the nitrogen mustard series and allied chemical substances. He has also discussed the possible mechanisms involved in the action of the drugs on cell constituents. It is suggested that the drugs of these series combine with what are called nucleophilic groups in proteins and nucleic acids and that the resulting structure has either an antitumour or a carcinogenic effect. Different drugs unite with different nucleophilic groups. Mustard gas by itself is much too dangerous to use and is not very effective, but by the introduction of other groups into the molecule or the replacement of chlorine by other elements or groups substances have been obtained which are much less toxic and much more active against malignant cells. Some of these substances such as TEM, triethylene melamine, and TEPA, triethylene phosphoramide, have been found very useful in the treatment of lymphadenoma and leuchæmia and in the control of pleural and peritoneal effusions in cases of multiple cancer of the pleura and peritoneum respectively. None of the drugs seem to have been very successful in the treatment of solid cancers.

Almost ten years ago Shay and his co-workers, using methylcholanthrene as a carcinogenic agent in an endeavour to produce gastric cancer in rats, failed in their attempt, but succeeded in producing instead adenocarcinoma of the breast and a variety of transferable leuchæmias. Therefore, while investigating the treatment of leuchæmia with triethylene thiophosphoramide, which is related to nitrogen mustard, they also tried it with good effect on two patients with recurrent adenocarcinoma of the breast. Following this up, J. C. Bateman[2] has described the results obtained in the treatment of very advanced cancers in various parts of the body by triethylene thiophosphoramide, which is TEPA with the oxygen replaced by sulphur. Ninety-nine patients with far advanced cancer were treated. Injections of the drug dissolved in water were given in a variety of ways—by the intravenous, intramuscular, intrapleural or intraperitoneal routes and into the mass of the tumour when this was superficial. The last method was the most successful and larger doses could be given. The majority of the patients were treated on an ambulatory basis at weekly intervals or longer when objective manifestations were minimal, but recurrences tended to take place if the intervals were longer than three weeks. Mammary and ovarian carcinoma appeared to be most sensitive to doses of triethylene thiophosphoramide tolerated by the human patient. Objective regression of the tumour, including decrease or disappearance of soft tissue masses, healing of ulcerations, recalcification of bone lesions and control of effusions, was noted in 28 of 34 patients with far advanced cancer of the breast. Similar results were obtained in 10 of 12 patients with ovarian cancer. Tumours of other parts of the body were much less sensitive to the drug, but even in these there was some palliation. The action was not rapid as it is in the treatment of lymphomata with nitrogen mustard, but was slow and progressive over several weeks. There were

[1] *J. Pharm. & Pharmacol.*, May, 1955.
[2] *New England J. Med.*, May 26, 1955.

no serious side effects, excepting sometimes a rapid fall in leucocytes which was the indication to stop treatment for a few weeks. The possibility that triethylene thiophosphoramide will cure any form of cancer is unlikely. However, it appears that the drug is a useful palliative agent, especially in the treatment of mammary and ovarian carcinoma.

J. C. Bateman *et alii* have tried the effects of triethylene thiophosphoramide in the control of neoplastic effusions.[1] The drug was injected in solution in water into the appropriate serous cavity. Control of pleural effusion lasting from one to nine months was achieved in 10 of 17 patients with mammary and ovarian cancer. Ascites from other types of tumours was not much affected. Response to therapy appears to be related to the primary site of the tumour responsible for the effusion.

Bergel points out that the beginning of effective chemotherapy in the field of cancer is not much older than one decade, and though the rate of advance cannot yet reach that in the treatment of infectious diseases, the prospects are far from being dull.

CONSTIPATION.

WETTING AGENTS have multifarious uses. Although one of them has been in use for the treatment of constipation for some fifteen years, the first report concerning it has only recently appeared. J. C. Wilson and D. A. Dickinson[2] state that dioctyl sodium sulphosuccinate, commercially known as "Aerosol O.T.", causes no intestinal irritation, does not interfere with normal bowel function and brings about no other evident ill effects. Large doses given to various animals for long periods caused no ill health, had no effect on red or white cells, and produced no changes apparent *post mortem*. Many hundreds of patients have been given it for the treatment of constipation without being harmed in any way. Absorption studies on infants gave no evidence that the drug caused either more or less absorption of food from the intestinal tract. In 1% aqueous solution its taste is not so bitter that it cannot be disguised easily, and added to infants' feeding bottles it is readily taken. Its most successful use was in patients with impacted fæces, most commonly in children with megacolon, to whom it was administered both by mouth and in enemas, in combination with mineral oil. Used thus, it softened fæcal matter too extensive and too hard for digital removal, by permitting water or the mineral oil to penetrate the mass, so that spontaneous evacuation became possible. The dosage used was dioctyl sodium sulphosuccinate, 1% aqueous solution, two millilitres three times daily by mouth in milk or fruit juice; five millilitres combined with 30 to 60 millilitres of sodium chloride solution or mineral oil as an enema. For infants with hard lumpy fæces causing anal fissure, 15 drops of the 1% solution given twice daily by mouth produced softened and easy motions. It has also served well bedridden, paralysed patients; adults need two millilitres, children one.

Undeniably it is a good thing that a method exists of securing evacuation of hard fæces based on penetrating and softening the mass with a wetting agent, instead of irritating the bowel wall with purgatives until it weeps enough fluid to soak the mass through. There are always occasions when prompt and innocuous treatment for fæcal impaction is needed. But it would be a far better thing if people could be educated to avoid constipation by natural means. Some general practitioners save themselves a lot of time and trouble by taking time to lecture their patients, whenever the occasion arises, on the following lines: "Your bowels are constructed to handle soft, mushy contents; you have abundant evidence, yourself, that they have great difficulty in handling hard, dry lumps. Now, there are two things necessary to keep your bowel contents soft and mushy. The first is some kind of fibrous matter to keep the contents crumbly—you can supply this by making sure that you eat at least one good helping of greens each day. The second is water. The fluid you drink is disposed of in three main ways: first, some is used for sweat, to keep your temperature normal; next, some is used by the kidneys to pass out waste materials as urine. What is left over remains in the bowel. If there is enough left over to keep the bowel contents soft and moist, the bowels can do their job easily; if there is not enough left over, they have to struggle with hard, dry masses. Every time you suffer from constipation, you will know that you did not drink enough fluids the day before." It might be advantageous if doctors could keep a supply of leaflets printed with something like that, and hand them out to suitable patients, together with the advice that they should try to form the habit of going to stool at a fixed time each day.

[1] *Arch. Int. Med.*, May, 1955.
[2] *J.A.M.A.*, May 28, 1955.

THE TREATMENT OF PREMATURE INFANTS.

THE commonly accepted treatment of premature infants is characterized by keeping them at an equable room temperature, oiling their bodies and keeping them warmly covered. They are not bathed. It should be noted that the temperature of most premature babies is subnormal. Some interest therefore attaches to a communication by G. Jobbich and H. Schaefer.[1] These authors state that the mortality of premature infants with subnormal temperatures is something like 30%. The treatment adopted by the authors has been based on experiences with artificial hibernation and also on the results of studies by Eckstin *et alii* which showed that the premature or newborn infant could stand subnormal temperatures better than the adult. Eckstin and his co-workers thought that the death of the infant occurred not so much because of cooling as because of warming up which was too rapid. Jobbich and Schaefer treated 45 premature newborn infants with subnormal temperatures in the following way. The temperature was recorded and the baby was wrapped in a blanket covered with a down quilt and again covered with a woollen blanket. No hot water bottles were used. The cradles were placed in a room which had an average temperature of 22° C. (71·6° F.). Every morning a bath was given, the water of which had a temperature of 35° C. (95° F.); under this treatment the temperature of the infants at the end of the third week approached the normal level, but the respiratory frequency was somewhat lower than that of infants who were not premature. In the feeding of these infants there was no essential difference from that of the normal infant. Jobbich and Schaefer claim that with this so-called cooling method the mortality of infants is only 15%.

HÆMATEMESIS.

THE diagnosis of the cause of hæmatemesis is very frequently in doubt. It is the custom to state that the hæmorrhage is due to cirrhosis of the liver if alcoholism is suspected, or peptic ulcer if there is any reason to suspect such a condition. In fact, however, these diagnoses are mere suppositions—in other words, guess work. If the patient does not die, the presence of cirrhosis of the liver cannot be proved, and the X-ray examination of the stomach made one to three weeks after the hæmorrhage frequently shows no abnormality. So, in a great proportion of cases the clinician is left in the dark as to the true diagnosis. In line with these thoughts is a discussion by D. C. Fainer and J. A. Halsted[2] of the sources of upper alimentary tract hæmorrhage associated with cirrhosis of the liver. Two hundred patients with a diagnosis of Laennec's cirrhosis were reviewed. Clinical, X-ray and gastroscopic evidence was considered. Hæmorrhage from

[1] *Deutsche med. Wchnschr.*, 1955, No. 2, page 73.
[2] *J.A.M.A.*, January 29, 1955.

the upper part of the alimentary tract had occurred in 38% of patients with a clinical diagnosis of Laennec's cirrhosis. Œsophageal and gastric varices were found to be responsible in 61% of cases. Peptic ulcer and gastric erosion were shown to be the cause in 23% and gastric and duodenal ulcers in 23%. More than one source of bleeding was found in 30% of cases. Fainer and Halsted consider that in view of the doubt as to diagnosis in many cases of hæmatemesis, treatment should consist of blood replacement and œsophageal tamponade. Ulcer management is indicated in view of the high incidence of peptic ulcer; administration of antacids or continuous gastric suction is suggested. If there is doubt as to the source of bleeding, emergency X-ray examination and endoscopy are recommended. If the diagnosis of peptic ulcer is fairly clear, immediate surgical intervention may be indicated. In this series emergency gastric resection was probably life-saving in three cases.

LIGAMENT PROLIFERATION BY THE INJECTION OF "SYLNASOL".

SODIUM PSYLLIATE has been used for twenty years to induce proliferation of fibrous tissue in the injection treatment of hernia. More recently the drug has been injected into stretched and weakened ligaments to cause them to become thicker and stronger, thus providing better support to joints. In a paper entitled "Joint Stabilization", G. S. Hackett and D. C. Henderson[1] present the results of their experiments on rabbits. They used the gastrocnemius and superficial flexor tendons, because joint ligaments are too small and inaccessible, but it must be agreed that any results obtained on tendons would be analogous to those on ligaments. "Sylnasol" was the proliferant used; an injection was given into the whole length of the tendons of one side, repeated after six weeks and again after fifteen weeks. The tendons of the opposite side were used as controls. Macroscopically and microscopically enlargement, thickening and increased fibrosis of the treated tendons were very apparent, and there was no evidence of necrosis or damage to nerves or blood vessels; increases in diameter up to 40% were secured.

Hackett has been using "Sylnasol" for years to stabilize the sacro-iliac joint. He points out that when ligaments become stretched, abnormal movement is possible, giving rise to pain; the injection of local anæsthetics relieves the pain but does nothing towards curing the condition. Various sclerosing agents have been used, but the rapid and extensive proliferation of fibrous tissue, with the absence of harmful effects now demonstrated conclusively for the first time, suggests that sodium psylliate is a satisfactory agent for this form of treatment.

CATARACT BEFORE THE AGE OF FIFTY YEARS.

THE onset of cataract in young adults is not common and is often associated with a generalized disorder. It is incumbent on the physician to be aware of such association and to be able to diagnose the presence of cataract, a finding which may confirm his diagnosis of some systemic disorder. In a long and thoughtful paper R. J. Meyer has made a medical classification of cataract before the age of fifty years and has discussed the various conditions in which cataract appears.[2]

Congenital cataract is divided into true developmental defects and environmental developmental defects. The former are usually bilateral and are stationary after birth. There is frequently a marked heredofamilial tendency and occasionally consanguinity is found. This cataract has been described as a single genetic defect transmitted as a dominant; the defect of the lens is probably related to defects in the germ plasma primarily. The environmental developmental cataracts are present at birth, but arise mainly from maternal influences; the most important single cause is rubella infection in the mother during the first three months of gestation. Other intrauterine infections which have been implicated in this type of cataract include maternal syphilis, mumps, morbilli, varicella, influenza and toxoplasmosis. Cataracts associated with genetic disorders are seen in Mongolism, Werners's and Rothmund's syndromes, myotonic dystrophy, inborn errors of metabolism as galactosæmia, Wilson's disease and organic acid urea. Of the endocrine diseases associated with cataract, cretinism and myxœdema, *diabetes mellitus* and hypoparathyroidism come immediately to the mind. Hypocalcæmia not associated with hypoparathyroidism is also a cause of cataract.

A rare disease associated with cataract is "Tahayasu's disease", a condition characterized by obstruction of the great vessels in the aortic arch, which produces a symptom complex of dimness of vision on arising or exercise, syncope, vertigo or light-headedness, headache, convulsions, aphasia and absence of radial pulses. The most common cause is syphilitic aortitis, and aneurysm need not be present.

Atopic dermatitis (neurodermatitis) is associated with cataract; the association of cataract in other skin disorders is probably a coincidence. Finally certain poisons such as dinitrophenol, ergot and naphthalene and physical agents such as direct trauma, radiant heat, X radiation, and atomic radiation may cause cataracts. Meyer concludes tht not only is knowledge of this type helpful in suggesting diagnosis, but also that early recognition of certain of these entities and promptness in treatment may prevent or reduce ophthalmic complications.

COMFORT IN SUMMER CLOTHING.

MANY tests have been devised for the evaluation of coolness in summer clothing, but most have been too gross for the detection of moderate variations between materials. B. L. Perti and B. B. Lal[1] have recently devised a test which gives a much finer distinction. Based on the principle that human comfort is related to skin temperature, it measures the drop in the temperature of the skin surface from a standard level following exercise. In the series of tests reported the subject was clothed in standard trousers and in a shirt of the material to be tested. He was placed in an air-conditioned room; the dry bulb temperature was 95° F. ± 1° and the relative humidity 60% ± 3; thermocouples were strapped to four points on his trunk. He then cycled on an ergometer for fifteen minutes, by which time his trunk was fully covered with sweat. The thermocouples were then read, after which a stream of air flowing at 300 feet per minute was directed onto his trunk, and temperatures were read each minute. In a series during which 60 readings were made on each of ten subjects, the fall in skin temperature after six minutes was as follows: subject nude, 2·7° F.; wearing an Indian muslin shirt (*kurta*), 4·0° F.; wearing a cellular cotton bush-shirt, 2·3° F.; wearing a British gaberdine jungle shirt, 1·8° F. These results were subjected to statistical analysis, and were found to be significant. This seems to be the most critical test so far devised, and although the variations between the types of materials tested are of the order which might be expected, it is most satisfactory to have a means of making accurate measurements. The authors do not offer any opinions as to why the nude subject did not cool so quickly as the one clothed in muslin, but it seems obvious that the thin material, by absorbing the sweat and distributing it more efficiently to the current of air, afforded better opportunities for evaporation than were presented by large drops of sweat on the bare skin surface. The thicker materials, while they would probably absorb the sweat equally well, would protect it from evaporation for a longer period.

[1] *Am. J. Surg.*, May, 1955.
[2] *New England J. Medicine*, 1955, 252: 622.

[1] *Nature*, May 28, 1955.

Abstracts from Medical Literature.

PHYSIOLOGY.

Factors Affecting Maximal Breathing Capacity.

B. M. LEWIS AND J. W. MORTON (*J. Appl. Physiol.*, November, 1954) report that the maximum breathing capacity is increased during the inhalation of 7·5% of carbon dioxide and during the hyperpnœa following exercise. It is increased by the injection of adrenaline but not by breath-holding. It is decreased by previous hyperventilation. Neither reflex drive to respiration nor "warming up" of the respiratory muscles can explain the increase in maximum breathing capacity. At similar minute volumes of ventilation, the subjects often noted dyspnœa when breathing 7·5% of carbon dioxide, but not during muscular exercise. The dyspnœa caused by carbon dioxide inhalation is still unexplained, but does not appear to be related to an increase in the work of breathing that is disproportionate to the minute volume of ventilation. Neither respiratory drive alone nor prior "warm-up" of the respiratory muscles alone seems to provide a satisfactory explanation of the increased maximum breathing capacity during exercise and carbon dioxide inhalation. Injection of adrenaline, however, produces a similar increase, and liberation of endogenous adrenaline may be an important factor in the increased maximum breathing capacity noted during inhalation of 7·5% of carbon dioxide and after muscular exercise. It is possible that the interaction of several factors, which individually are ineffective, may, by potentiation, lead to an increase in maximum breathing capacity. Such a schema, however, is purely conceptual, and other factors as yet unidentified may be of more importance.

Induced Hypotension.

S. H. NGAI AND E. C. NELSON (*J. Appl. Physiol.*, September, 1954) state that hexamethonium bromide and other ganglionic blocking agents have been used recently during anæsthesia to induce hypotension for the purpose of reducing bleeding from the operative site. This induced hypotension may be necessary when lesions are very vascular; but if it is employed indiscriminately, death or permanent ischæmic damage may result. The blood flow through the internal carotid and occipital arteries and the arterial pressure were measured continuously during hexamethonium-induced and d-tubocurarine-induced hypotension in dogs. In 11 of 14 animals studied, the rate of blood flow was a direct function of the arterial pressure. In six animals there was evidence of compensatory vascular relaxation during the induced hypotension.

Alveolar Ventilation at Very Low Tidal Volumes.

W. A. BRISCOE, R. E. FORSTER AND J. H. COMROE, JUNIOR (*J. Appl. Physiol.*, July, 1954) report that after inspiration of as little as 60 millilitres of a mixture of 80% helium and 20% oxygen, there is an appreciable quantity of helium in the expired alveolar gas. This means either that the dead space must be very small (of the order of 50 millilitres) during small breaths, or that during small inspirations inspired air penetrates the dead space into the alveoli without washing all the dead space gas into the alveoli. The second explanation is probably the correct one. Therefore, with very low tidal volumes the formula (alveolar ventilation equals tidal volume *minus* dead space volume) is incorrect; it becomes correct only when the tidal volume is large enough to flush completely the dead space. This may explain the clinical fact that some patients can live despite the fact that they are breathing very small tidal volumes. These findings are important in a complete evaluation of hypoventilation in controlled and artificial respiration.

Determination of Human Body Surface Area from Height and Weight.

J. SENDROY, JUNIOR, AND L. P. CECCHINI (*J. Appl. Physiol.*, July, 1954) report that a simple, rapid and accurate method has been developed of estimating human body surface area from height and weight. From the empirical relationships of height *plus* weight (in centimetres and kilograms respectively) and from the "shape" factor of the ratio of weight to height, a chart has been constructed for the graphical estimation of surface area values in the range from 0·05 to 3·0 square metres. From this master chart a diagram has been constructed from which surface areas may be obtained with the same accuracy as and more conveniently than from values of height and weight alone. A comparative evaluation and a statistical analysis have been made of the method applied to 252 measurements of surface area. The results indicate a margin of superiority in respect to accuracy, especially in the case of abnormal body types, for the present graphical method, as compared with the well-known Du Bois height-weight formula. The self-adjusting power equation of Boyd has been found to give results generally comparable to those obtained with the authors' diagram. According to these tests, the equation of Breitmann has been found to be biased and not sufficiently accurate to merit consideration for further use. All factors considered, the presently proposed graphical method would seem to be generally superior to other methods of obtaining a value for surface area from the simple physical measurements of height and weight. A consideration of the rationale of the anthropometric relationships involved indicates that they are in accord with accepted concepts pertaining to the growth and development of the human body.

Gastric Contents, Mental Concentration and Production Rate.

R. C. HUTCHINSON (*J. Appl. Physiol.*, September, 1954) reports that in two ninety-second concentration tests administered in the late hours of the morning to 38 subjects, better results were obtained by those subjects who first consumed a simple light refreshment consisting of sandwiches and tea, the difference for one of the tests being significant at the 1% level. In a three-minute concentration test administered in the late hours of the morning to 88 subjects, better results were obtained by those subjects who first consumed a simple light refreshment consisting of sandwiches and fruit cordial, the difference being significant at the 1% level. In fifteen-minute concentration tests administered to 10 senior typists who on five consecutive days consumed meals the solid portions of which varied from six to eighteen ounces, the work was performed best after a meal the solid portion of which weighed nine to twelve ounces, the difference being significant at the 1% level. In fifteen-minute typing tests administered to 40 junior typists who on five consecutive days consumed meals the solid portions of which varied from eight to twenty-four ounces, the effect of the size of meal could be rendered insignificant if motivation to do well was sufficiently great. It is concluded, therefore, that the mid-morning consumption of small amounts of solid food may increase ability to concentrate from the time the food is consumed until lunch time, and for those at work an increase in production rate will almost certainly result. On the other hand, if the consumption of solid food exceeds a certain amount, which could be in the vicinity of nine to twelve ounces for those engaged in sedentary work, there may be a decrease in production rate, and this places limitations on the value of a full three-course meal consumed at any time during the working day.

BIOCHEMISTRY.

ACTH.

D. STONE AND O. HECHTER (*Arch. Biochem.*, August, 1954) have perfused cow adrenal glands with C^{14}-labelled acetate, cholesterol and progesterone in the presence of and absence of added ACTH. The data demonstrate that ACTH greatly increases the incorporation of C^{14} into corticoid only when cholesterol is used as a precursor. The combined results strongly indicate that the major in-vitro effect of ACTH is concerned with a step involved in the conversion of cholesterol to progesterone. The studies with acetate-C^{14} demonstrate a pathway of corticoid biosynthesis which is not significantly influenced by ACTH, and which does not involve the formation of cholesterol as an obligatory intermediary.

Amino Acids.

W. H. STEIN AND S. MOORE (*J. Biol. Chem.*, December, 1954) have shown that deproteinized human blood plasma contains 28 ninhydrin positive compounds identifiable by chromatography on columns of Cowese 50-X4. The results, taken in conjunction with the previous values for glutamine obtained by Hamilton and Archibald, account for 95% to 100% of the individual amino acids which contribute to the -amino N of post-absorptive plasma (4·1 milli-

grammes per 100 millilitres. In agreement with previous investigation, no detectable quantities of peptides have been found in venous plasma of subjects either in the fasting state or after a protein meal.

Mucoproteins.

A. J. ANDERSON AND N. MACLAGAN (*Biochem. J.*, April, 1955) have investigated a method of isolating mucoproteins from normal male urine by the use of adsorption on benzoic acid. Two similar mucoprotein preparations were obtained in a yield of approximately 33 milligrammes per litre of urine. A colorimetric method is described for estimation of urinary mucoproteins in normal and pathological urine. No significant diurnal excretory rhythm was observed in four normal subjects studied. Observations over a period of five months on one normal subject are recorded. Ten normal male subjects excreted $146 \pm 7{\cdot}5$ milligrammes of mucoprotein in twenty-four hours, while ten normal females excreted $106 \pm 6{\cdot}4$ milligrammes. The sex difference was statistically significant ($40 \pm 9{\cdot}8$ milligrammes).

Fœtor Hepaticus.

F. CHALLENGER AND J. M. WALSHE (*Biochem. J.*, March, 1955) have studied a case of massive hepatic necrosis in which *fœtor hepaticus* was present in the patient's breath and a similar odour in the urine. Passage of nitrogen through the urine into mercuric cyanide yielded mercury dimethyl mercaptide ($Hg(SCH_3)_2$). The probable source of the methyl mercaptan is the CH_3S- group of methionine, the normal demethylating processes being inhibited by liver damage. Both methyl mercaptan and dimethyl disulphide may be exhaled in the breath. Analogous reactions in certain micro-organisms, and with the enzyme thionase, and the toxicity of methionine to man and animals are considered.

Alcohol.

J. J. VITALE *et alii* (*J. Biol. Chem.*, October, 1954) have shown that in normal rats the administration of pyruvate or acetate inhibits the oxidation of alcohol as measured by the excretion of $C^{14}O_2$ after the administration of ethanol-1-C^{14}. Since the disappearance of alcohol from the blood is also delayed, the inhibition is apparently at the first stage of alcohol oxidation to acetaldehyde. The administration of large amounts of niacin prior to study prevents the inhibitory effect of pyruvate. In thiamine deficiency the pyruvate inhibition is decreased. It is suggested that the inhibition is due to competition for the coenzyme DPN, this coenzyme being preferentially reduced in the tricarboxylic acid cycle rather than by alcohol oxidation when it is available in limited amounts. Limited studies upon alcohol disappearance from the blood of human subjects suggest a similar effect.

"Antabuse"

A. W. WASE AND J. CHRISTENSON (*J. Biol. Chem.*, November, 1954) have studied the antithyroid action of "Antabuse". It was found that "Antabuse" reduced the thyroid I^{131}-trapping activity of the rat to 44·8% of that of control animals. "Antabuse" reacts with iodine to form a complex substance. Iodine is reduced by "Antabuse" in an aqueous medium.

Protein Synthesis.

J. SUTMAN *et alii* (*J. Biol. Chem.*, January, 1955) have studied the effect of changing the protein content of the diet, either direct or indirect, on the ability of slices from the livers of rats to incorporate labelled methionine (synthesize protein). They state that the effect is very significant; the highest rate of incorporation occurs with a 45% casein diet and the lowest with a protein-free diet. The rate with the high protein diet is twice that with the protein-free diet. On the basis of the incorporation observed either per unit weight of animal at the time of sacrifice or per unit weight of liver, the effect of fasting on the rat previously receiving a low protein or protein-free diet is to increase the rate (after one day). In contrast, the fasting of rats previously fed a diet of higher protein content results in a reduction in the rate. After four days of fasting, whatever the previous diet, the incorporation rate is seriously reduced from the level observed in the fed animal. The relationships between these observations and the replacement rates observed for plasma protein and for albumin *in vivo* are discussed.

Pituitary Enzymes.

J. B. MELCHIOR AND D. M. HILKER (*J. Biol. Chem.*, January, 1955) have found that the endogenous respiration of intact pituitary tissue obtained from albino rats was significantly higher than that of liver slices and was unaffected by addition of glucose or succinate. Enzymes of the tricarboxylic acid cycle were uniformly low in this tissue. Pituitary preparations failed to oxidize glycine, alanine, D-glutamate or L-glutamate. Glutamic-oxalacetic transamination was actively catalysed by pituitary preparations.

Lymphosarcoma.

S. KIT (*J. Biol. Chem.*, January, 1955) found that cell suspensions of the Gardner lymphosarcoma converted acetate-2-C^{14} to glycine-2-C^{14}. Total radioactivity due to glycine was as great as that due to the dicarboxylic amino acids. The conversion was also studied with mouse kidney, diaphragm, liver and hepatoma slices, and with rabbit appendix, rat thymus, mouse spleen, Ehrlich carcinoma and Mecca lymphosarcoma cells. Total radioactivity due to Gardner tumour glycine exceeded that of the other tissues by a factor of eight or more, while the specific activity was two or more times that of the other tissues. Although not unique to the Gardner tumour, this metabolic pathway would appear to be of greater metabolic significance in that tissue.

Multiple Myeloma.

F. W. PUTNAM AND S. HARDY (*J. Biol. Chem.*, January, 1955) have studied the rate of protein synthesis by the use of glycine in a patient with multiple myeloma who had an abnormal serum globulin and excreted a Bence-Jones protein. The synthesis of the two abnormal proteins appeared to be an independent process. The authors state that the Bence-Jones protein is rapidly excreted and is apparently derived directly from the nitrogen pool rather than via any plasma or tissue protein precursor. Protein synthesis has also been studied in a human subject with a plasma cryoglobulin who excreted a Bence-Jones protein. No evidence was found for the origin of the urinary protein from serum proteins or from any precursor relationship between the two abnormal proteins. The parallel decline in N^{15} in the glycine of the urinary protein and in the urinary urea, ammonia and total nitrogen indicates a direct interaction with the metabolic pool of nitrogen in the synthesis of Bence-Jones protein rather than the intervention of serum or tissue protein precursors. The renal excretion of both non-protein nitrogen and protein was retarded, probably owing to kidney damage.

Nephrosis.

D. L. DRABKIN AND J. B. MARSH (*J. Biol. Chem.*, February, 1955) have investigated the protein and carbohydrate metabolism in the experimental nephrotic state produced by the injection of rabbit anti-rat kidney serum into rats. After injection of glycine-2-C^{14}, serum, liver, kidney and urinary protein, but not heart protein, were more highly labelled in the nephrotic rats. In addition the rate of decline in the specific activity of these proteins was accelerated in the nephrotic rats. These findings indicate an increased turnover of body protein in nephrosis. *In vitro*, liver slices from the nephrotic rats showed an increased incorporation of labelled glycine into the proteins and an increased oxidation of glycine to carbon dioxide, indicating an acceleration of amino acid metabolism. The experimental nephrotic state was found also to be accompanied by a marked decrease in the level of liver glycogen. Fasting hypoglycæmia was demonstrated. It was found also that the incorporation of acetate-1-C^{14} into cholesterol and fatty acids *in vitro* was greatly diminished in nephrotic rat liver and unchanged in nephrotic rat kidney. In the case of cholesterol, the liver of the nephrotic rat responds to the high level of serum cholesterol by a diminution in the rate of cholesterol synthesis. This behaviour appears to be analogous to that previously observed with the feeding of cholesterol. The diminished incorporation of acetate into liver fatty acids is related to the low level of liver glycogen in the nephrotic rat. Direct evidence for mobilization of fat and cholesterol was obtained by the estimation of total body fatty acids and cholesterol. The cholesterol content of the blood, liver and kidney of the nephrotic rats was increased, and a corresponding decrease in cholesterol was found in the remainder of the body. A 37% reduction in total body fatty acids indicates that the hyperlipæmia of the nephrotic rat is essentially a mobilization lipidæmia. Fat is transported from body stores to be used for energy.

British Medical Association News.

ANNUAL MEETING.

THE annual meeting of the Western Australian Branch of the British Medical Association was held at Tintern Lodge, King's Park Road, Perth, on March 19, 1955, DR. A. L. DAWKINS, the Acting President, in the chair.

MINUTES.

The minutes of the annual meeting held on March 13, 1954, had been printed and circulated amongst members. They were taken as read and adopted on the motion of Dr. R. G. Linton, seconded by Dr. H. G. Dicks.

PRESIDENT'S ADDRESS.

In the absence in Europe of the President, Dr. J. H. Stubbe, his President's Address was read by Dr. A. L. Dawkins, Acting President (see page 185).

ANNUAL REPORT OF THE COUNCIL.

The annual report of the Council for the year ended March 19, 1955, was received and adopted. The report is as follows.

The President and members of the Council of the British Medical Association (Western Australian Branch) have much pleasure in presenting the fifty-sixth annual report of the Branch for the year ending March 19, 1955.

Membership.

The membership of the Branch has increased during the twelve months ended December 31, 1954, by a net amount of seven as follows. Gains were: new members 26, transfers from other Branches 35, total 61. Losses were: transfers from this Branch 44, resignations 6, membership lapsed 1, deaths 3, total 54. Membership of the Branch totals 529.

Obituary.

With deep regret we record the deaths of the following members which occurred during the year: Dr. G. A. Murray, Dr. C. E. Young and Dr. N. S. Williams. The sincere sympathy of the Branch is extended to the families of these late members.

Meetings.

In addition to the annual meeting seven general meetings of the Branch were held. The subjects of these meetings were as follows: April: Clinical demonstrations arranged by Dr. J. Stokes on behalf of the staff of the Royal Perth Hospital. May: "An Approach to the Treatment of Essential Hypertension: Two Opposing Points of View", Dr. J. P. Ainslie and Dr. C. Fortune. June: Combined medico-legal meeting. July: "The Clinical Uses of ACTH and Cortisone", Professor E. B. Astwood. August: "The Emotional Problems of Childhood", Dr. W. Wyatt. September: "The Treatment of Peripheral Vascular Disease", Sir James Learmonth. October: "The Use and Abuse of Antibiotics in Respiratory Disease", Dr. Scadding.

Council Meetings.

Fourteen meetings of the Branch Council were held. The record of attendance is as follows:

Dr. J. H. Stubbe (President)	12
Dr. C. W. Anderson (Honorary Secretary and Federal Representative)	14
Dr. A. R. Bean (Councillor)	12
Dr. B. O. Bladen (Chairman, Contract Practice)	8
Dr. B. W. Buttsworth (Vice-President)	13
Dr. F. W. Carter (Permanent Vice-President)	2
Dr. D. M. Clement (Assistant Honorary Secretary)	11
Dr. B. C. Cohen (Councillor)	13
Dr. H. L. Cook (Special Coopted Councillor)	11
Dr. D. E. Copping (Federal Representative)	11
Dr. S. E. Craig (Councillor)	5
Dr. A. L. Dawkins (Past President)	13
Dr. L. I. Henzell (Commissioner of Public Health)	7
Dr. W. S. Davidson (Deputy Commissioner of Public Health, deputizing for Dr. Henzell)	2
Dr. D. D. Keall (Honorary Treasurer)	12
Dr. R. S. W. Thomas (Councillor)	13
Dr. A. B. Wilson (Councillor)	14

Office-Bearers and Councillors.

The following members have been elected as office-bearers for 1955: *President*, Dr. B. W. Buttsworth; *Vice-President*, Dr. H. L. Cook; *Honorary Treasurer*, Dr. D. D. Keall; *Honorary Secretary*, Dr. C. W. Anderson; *Honorary Assistant Secretary*, Dr. D. M. Clement; *Chairman of Convocation*, Dr. A. L. Dawkins.

The permanent Vice-President of the Branch is Dr. F. W. Carter, who was elected to that position by resolution at the general meeting of June 17, 1953.

The following members have been elected as the five ordinary members of Council for 1955: Dr. A. R. Bean, Dr. S. E. Craig, Dr. D. W. Fleming, Dr. H. K. Pawsey, Dr. A. B. Wilson.

At this juncture it is desired to express on behalf of the Association the warmest appreciation of the work done by the retiring councillors.

Federal Council.

Dr. Colin Anderson and Dr. Donald Copping represented the Branch on the Federal Council during the past year, and it is desired to convey to them the Association's gratification of their strenuous efforts through a very tiring year.

A special general meeting was held at the Royal Perth Hospital on October 19 and was addressed by Dr. John Hunter, the General Secretary of the Federal Council, who spoke on problems arising out of the National Health Service.

Library.

The library has continued to operate in its customary efficient manner, and the Association is fortunate in having Dr. Ernest Beech as the chairman of its Library Committee. The general activities of the library will be the subject of a separate report.

Representation.

The Branch was represented by the following members at various meetings and conferences during 1954 as follows: Council of the British Medical Association, Dr. Miles Formby; Federal Council of the British Medical Association in Australia, Dr. C. W. Anderson, Dr. D. E. Copping; University Advisory Board in Medicine, Dr. H. H. Stewart; Australasian Medical Publishing Company, Limited, Dr. F. W. Carter; THE MEDICAL JOURNAL OF AUSTRALIA (Editor's representative), Dr. R. L. Leedman; State Health Council, Dr. D. M. Clement, Dr. H. L. Cook, Dr. A. B. Webster, Dr. M. F. Williams; Nurses' Registration Board. Dr. J. A. Love, Dr. L. E. LeSouef; Dental Board, Dr. I. O. Thorburn; College of Dental Science, Dr. I. O. Thorburn; Workers' Compensation Act Underwriters' Joint Committee, Dr. N. H. Robinson, Dr. H. M. Hill, Dr. G. B. Maitland; Saint John Ambulance Association, Dr. A. L. Dawkins; Optometrists' Registration Board, Dr. D. D. Paton; Sex Education (Parents and Citizens' Association), Dr. I. O. Thorburn; Protection of Practices, Dr. F. W. Carter; National Health Service Disciplinary Committee, Dr. F. W. Carter, Dr. H. L. Cook, Dr. H. J. Gray, Dr. C. W. Anderson.

Public Health.

We wish to take this opportunity of thanking Dr. Linley Henzell, Commissioner of Public Health, and Dr. W. S. Davidson, Deputy Commissioner of Public Health, for their cooperation and assistance during the year, which has been invaluable to the profession.

Australasian Medical Publishing Company, Limited.

Dr. F. W. Carter has continued with zeal to act as the nominated director of the company for this State and, as usual, has on several occasions kept the Branch Council fully informed with most comprehensive reports of directors' meetings.

Post-Graduate Committee.

The Post-Graduate Committee, under the very capable chairmanship of Dr. Dixie Clement, has had another very successful year, and its activities are the subject of a separate report presented by its honorary secretary, Dr. Adrian Lamb.

Contract Practice.

Again during 1954 the Contract Practice Committee has had to do considerably less work than usual, but such as was necessary has been carried out with the customary exactitude.

Social.

Medico-Dental Golf.—The medico-dental golf competition held at Karrinyup on July 21 was a great success. Forty players participated. The trophies, which were donated by F. H. Faulding and Company, were won by Dr. Craddock and Dr. Buttsworth. This is intended to be an annual event, and a shield which was donated first in 1936 has now been unearthed and will be played for each year. This year it was won by the dentists.

Medico-Legal Golf.—The medico-legal golf match and dinner was held at Royal Perth Golf Club on Wednesday, October 13. There were 44 players. The match resulted in a win for the medical profession. The trophy for the best four-ball score was won by Dr. Barnard and Dr. Kermode. The B.M.A.C. Trophy for the best individual score was won by the Chief Justice, Sir John Dwyer, with a score of two up. The dinner which followed in the evening was an outstanding success.

As both matches were particularly successful it is hoped that they can continue to be annual events.

Convocation.

Two meetings of convocation were held during the year, and as usual the main items for discussion were of a medico-political nature.

Staff.

The Council wishes to express to the office and library staffs its sincere thanks for their cooperation and help to the Association during the year.

Medical Benevolent Association of Western Australia.

The annual report of the Medical Benevolent Association of Western Australia was adopted on the motion of Dr. D. M. Clement, seconded by Dr. A. L. Dawkins.

The Committee of the Medical Benevolent Association of Western Australia has pleasure in presenting a statement indicating the receipts and disposal of funds during the past year and the financial position of the Association as at December 31, 1954.

Income was derived from subscriptions through the Western Australian Branch of the British Medical Association and from interest on Commonwealth loans and our Savings Bank account.

Total expenditure was in the nature of benevolent contributions to the welfare of the widows of six doctors.

Our invested funds remain the same at £1700, which together with bank balance of £1142 0s. 9d. totals £2842 0s. 9d.

The committee would express their sincere appreciation for the continued support of your Association.

Report of the Library Committee.

The report of the Library Committee for the twelve months ended March 15, 1955, was presented by Dr. Ernest Beech. It was adopted on the motion of Dr. R. G. Linton, seconded by Dr. D. D. Keall.

We beg to submit to you the report of the committee on the British Medical Association Library for the twelve months ended March 15, 1955.

The committee met on eleven occasions during the year and a report on library matters was submitted to Council each month.

Approximately three thousand requests for information were received personally and by telephone and letter. Five hundred and sixty-four books were borrowed as well as 2796 journals. We still continue to borrow rather heavily from other libraries who are always most cooperative. Approximately 90 books and journals were borrowed from the Branch libraries in the eastern States and from the Public and University Libraries. A special mention must be made, too, of the splendid and generously given help by the librarian of our own Public Health Department.

With the establishment of the Central Medical Library attached to the University of Melbourne, which concerns itself with collecting and distributing duplicate journals, we have been fortunate in filling in many gaps in our sets which should help reduce our interstate borrowings.

Fifty-nine books have been added to the library—nineteen bought by the library and 40 donated by THE MEDICAL JOURNAL OF AUSTRALIA, and private members. The small number of books actually bought is accounted for by the fact that at least half of them cost anything from six to ten pounds each and the money allotted for their purchase disappeared in an alarmingly short time. The Clinical Research Unit have added 70 books to their section, many of which have been borrowed by members during the year.

Our thanks are once again extended to those members who donated new and not so new, but just as valuable, books and monographs to the library.

Last year some individual sections of the profession donated the subscriptions to their own particular journals, and for this help the Library Committee was extremely grateful. It is hoped that they may feel inclined to continue their subscriptions for another year, and the money thus saved could be put, perhaps, towards buying some much needed books.

Organization of library material and library methods for the greater convenience of the librarian and members has been continually kept in mind, and numerous small changes have been put into effect. Some of these are:

1. Journal borrowing records. The rather clumsy method of record keeping used hitherto has been done away with, and a system of individual cards for each journal has been introduced, which makes tracing the whereabouts of any particular journal a very rapid process.

2. As the library has not sufficient funds to provide two copies of important reference books it was thought essential that these should not be absent from the library for more than seven days at a time at least. Accordingly they were

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Medical Benevolent Association of Western Australia.

Statement of Recepts and Payments for Year Ending December 31, 1954.

Receipts.	£ s. d.	£ s. d.
January, 1954.		
Funds on Hand:		
Commonwealth Savings Bank ..	1,079 16 11	
Commonwealth Treasury Bonds	1,700 0 0	
		2,779 16 11
Subscriptions through British Medical Association, Western Australian Branch:		
4th Quarter, 1953	10 10 0	
1st Quarter, 1954	391 13 0	
2nd Quarter, 1954	41 9 6	
3rd Quarter, 1954	13 13 0	
		457 5 6
Interest:		
Commonwealth Treasury Bonds	49 6 3	
Commonwealth Savings Bank ..	18 12 1	
		67 18 4
Bequest, Estate of the late Dr. K. G. Aberdeen		50 0 0
		£3,355 0 9

Payments.	£ s. d.	£ s. d.
December, 1954.		
Payment to Beneficiaries:		
Mrs. "A"	78 0 0	
Mrs. "B"	78 0 0	
Mrs. "C"	78 0 0	
Mrs. "D"	78 0 0	
Mrs. "E"	78 0 0	
Mrs. "F"	65 0 0	
		455 0 0
Institute for the Physically Handicapped		55 0 0
General Expenses:		
Cheque Book	2 0 0	
Bank Charges	1 0 0	
		3 0 0
Funds on Hand:		
Bank of New South Wales ..	1,142 0 9	
Commonwealth Treasury Bonds	1,700 0 0	
		2,842 0 9
		£3,355 0 9

marked with a sticker requesting their return in seven days and on the whole members have responded very well.

3. The Library Committee invited the cooperation of one or two representatives from each branch of medicine in reviewing the present library stock, in order that they might make suggestions as to what might be discarded as being of no further value either contemporarily or historically, and also what should be added to make each section as complete as possible. The members asked kindly gave of their time and energy, and as a result a lot of dead wood was cleared out and some valuable additions made.

4. A suggestion from a member of the hospital research staff was adopted, and new journals are now displayed in the library for one week for all members to read before being removed by any one member.

5. A typewritten catalogue of the newer books in the library has been, at last, accomplished, and kindly duplicated for us by the Branch office. This was done primarily for country members in an effort to give them any additional aids which can be devised in order to make up, in part, for the disadvantage they are at in being so far removed from actual access to the library.

6. Recently the shelves in the stacks have been numbered, and a typewritten guide to where each set of journals is stored has been attached to the outside. This will, we hope, particularly help those members who wish to use the library after hours.

The historical section of the library has evidently captured the interest of members, and many interesting books—and in one case an instrument—have been generously donated. In fact there is now not sufficient space for everything to be suitably displayed, and a larger portion of the library space will have to be allotted to this section in future if it continues to increase at this rate—which, of course, we hope it will.

At the last meeting we mentioned that the committee had asked Council's approval for the establishment of a Harry Lucraft Memorial Fund. This was duly given and up to the present members have contributed £22 1s. When there is enough in the fund a suitable piece of furniture and a plaque will be bought and put in the new library quarters when these are established.

Just what form these will take and where they will be situated is not yet certain. The foundation of a central library which would serve the British Medical Association, Medical School and general hospital services would be ideal, thereby eliminating the small individual libraries, and preventing reduplication. The British Medical Association Library Committee regret that so far, although they have asked for information, no meeting of the institutions concerned has yet been held so that some concrete plans can be laid down.

Once again the committee acknowledges with many thanks its indebtedness to the Medical Board. The grant for 1954 was seven hundred and fifty pounds (£750), but owing to a credit balance of £13 13s. 9d. we started the year with £763 13s. 9d. The money was distributed as follows: books, £92 18s. 6d.; journals and Year Books and indices, £468 16s.; binding, £198 10s. 9d.; leaving a credit balance of £3 8s. 6d.

Finally we would like to express once again our gratitude to the library staff for their work during the year. At the beginning of this year Miss Clarke left to take up a position as full-time assistant to the Clinical Research Unit, but the library now has Miss Robinson, who has settled into her work in a surprisingly short time and gives much valuable help to the librarian.

Report of the Honorary Treasurer.

The report of the Honorary Treasurer and the financial statement for the year ended December 31, 1954, were presented by Dr. D. D. Keall, Honorary Treasurer. The report was adopted on the motion of Dr. H. J. Gray, seconded by Dr. D. Copping.

I am glad to be able to report the financial state of the Branch is very satisfactory, and that the auditors' report

British Medical Association (Western Australian Branch).

Statement of Income and Expenditure during Twelve Months Ended December 31, 1954.

Income.

	£ s. d.	£ s. d.
By Members' Subscriptions		6,827 2 0
„ Interest:		
(a) Commonwealth Bonds	83 9 4	
(b) Australasian Medical Publishing Company, Limited—		
(i) Debentures "A"-"D"	17 5 0	
(ii) Debentures "E"	44 7 4	
		145 1 8
„ Sale of Lists		10 0
„ B.M.A.C.—Revenue Account		280 18 6
		£7,253 12 2

Expenditure.

	£ s. d.	£ s. d.	£ s. d.
To Subscriptions to:			
(a) Members' Journals:			
(i) Aust. Med. Pub. Co., Ltd.	£534 0 0		
Less Series "E" Debentures	£267 0 0		
	267 0 0		
(ii) B.M.A., London	845 3 6		
		1,112 3 6	
(b) Federal Council, Sydney		548 2 0	
(c) Medical Benevolent Association		473 0 6	
(d) Flying Doctor Service		1 1 9	
(e) Historical Society of Western Australia		12 6	
		2,134 19 6	
„ Cost of Administration:			
Advertising	8 8 0		
Audit Expenses	31 10 0		
Depreciation	50 9 0		
Duplicating	159 3 7		
General Expenses	42 15 1		
Legal Expenses	21 19 6		
Library Expenses	170 6 11		
Medico-Political	27 0 2		
Postages and Petty Cash	233 9 11		
Printing	4 3 5		
Rent	370 5 4		
Salaries:			
Secretary	£702 0 0		
Typists	£594 14 11		
Librarians	£755 4 0		
Clerical Assistant	£144 1 0		
	2,195 19 11		
Stationery	69 8 3		
Telephone	94 17 1		
		3,479 16 2	
		5,614 15 8	
„ Excess of Income over Expenditure		1,638 16 6	
		£7,253 12 2	

(which has been circulated) shows that the income for the year exceeded expenditure by £1638.

When compared with the previous year's balance sheet, certain features are of interest. Income increased by slightly less than £300, this being accounted for by an increase in membership. Expenditure was reduced by about £250, despite an increase of £100 in our rent. The main savings were in salaries—caused by a reduction in the junior secretarial staff in the library and the Association office. Part of this saving, unfortunately, will not continue. Expenditure in the medico-political field was substantially less.

The purchase of Clarendon House by BMAC for the Association has, as yet, had very little impact on our finances, as the assistance by members with free loans has kept the overdraft and interest rates small. There is to be a separate report on Clarendon House, but I feel that it is not out of place for the Treasurer to express the Association's gratitude to members who have responded to the appeal, and have made gifts and loans. We are steadily acquiring a sizeable equity in the building, and can look forward, if members continue to support their Association, to attaining ownership of our own house in a remarkably painless fashion. However, I must urge members not to feel that the job is done, for unless these loans continue, we (through BMAC) will be faced with heavy expenditure in paying interest. The system of free loans, which has been successful, is to my mind a most intelligent way of coping with the problem, and the response reflects great credit on our members. The gift aggregating £583 to the Building Fund, which is shown in the balance sheet, is acknowledged with gratitude.

It is thought that it may be of some interest, if we dissect the expenditure, to show in round figures how each subscription is spent. There are certain fixed charges: THE MEDICAL JOURNAL OF AUSTRALIA, £1; *British Medical Journal*, £1 10s.; Federal Council, £1; Medical Benevolent Fund, £1. The library takes something in the region of £2, salaries about £2 10s., and rent and administration rather more than £2. In addition, of course, the Association gets a tremendous amount of voluntary work done by certain members of Council. I feel that I can speak freely, as the Treasurer's job is not a particularly arduous one, and would like to bring to your notice the very large amount of work in the affairs of the Association which is done by certain members.

The subscription was increased two years ago by £3 3s. a head. At that time costs were rising rapidly, and the Association's finances were in a very difficult state. In the last two years our finances have improved, and we have shown a reasonable surplus. This matter was discussed at the last Council meeting, and it was agreed that a reduction in subscription is now possible. It is proposed to ask a later general meeting to sanction a reduction of £1 1s. to take effect next year.

REPORT OF THE POST-GRADUATE COMMITTEE.

The report of the Post-Graduate Committee of the Branch for 1954 was presented by Dr. Adrian Lamb. It was adopted on the motion of Dr. R. McKellar-Hall, seconded by Dr. Bean.

The function of this committee is to promote and organize post-graduate medical teaching in Western Australia on behalf of the British Medical Association. This teaching must be directed to the recent graduate, the general practitioner and the specialist, and must reach out to the academically isolated doctors.

This committee has tried to fulfil its functions during the past twelve months in the following ways: (*a*) by invitation to overseas lecturers; (*b*) by invitation to distinguished Australian teachers; (c) by encouragement of local talent, where possible young local talent; (*d*) by sending teams of lecturers to country districts; (*e*) by recording suitable lectures and making the tape recordings available; (*f*) by running refresher courses; (*g*) by organizing a full course in anatomy and physiology for the primary F.R.A.C.S.; (*h*) by the sponsorship of doctors wishing to do post-graduate work overseas; (i) by arranging a post-graduate week; (*j*) by arranging the clinical aspect of each British Medical Association monthly meeting.

The following overseas speakers addressed the profession: Professor Astwood and Professor Patten from the United States, Professor Gordon King from Hong Kong, Dr. Scadding and Sir Macfarlane Burnet from England, and

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BRITISH MEDICAL ASSOCIATION (WESTERN AUSTRALIAN BRANCH).

Statement of Assets and Liabilities at December 31, 1954.

ASSETS.	£ s. d.	£ s. d.
Subscriptions in Arrears		130 4 0
Sundry Funds Overdrawn:		
C.R.U. Grant	33 13 3	
Post-graduate Grant	45 1 5	
		78 14 8
Investments:		
Australasian Medical Publishing Company, Limited—		
Debentures "A"-"D"	345 0 0	
Debentures "E"	1,442 12 6	
	1,787 12 6	
Commonwealth Bonds	1,410 0 0	
British Medical Agency Shares	9 0 0	
		3,206 12 6
Fixed Assets:		
British Medical Agency—B.M.A. House Loan Account	15,636 7 1	
Furniture and Fittings at 1.1.54 £863 11 10		
Since bought £145 12 0		
£1,009 3 10		
Less Depreciation £50 9 0		
	958 14 10	
		16,595 1 11
		£20,010 13 1

LIABILITIES.	£ s. d.	£ s. d.
Subscriptions in Advance		66 3 0
Sundry Funds in Credit:		
B.M.A. House—Members' Loans	9,280 0 0	
Building Fund	758 19 0	
Car Badge Account	50 8 8	
Entertainment Account	*3 17 3*	
Library Donations	15 11 2	
Cyril Bryan Fund	202 8 10	
Harry Lucraft Fund	*22 1 0*	
Medical Board Grant	3 8 6	
Medico-Legal Golf	9 1 6	
Nelson Bequest	17 7 1	
Post-Graduate Committee	*140 14 0*	
Publicity	147 12 0	
		10,651 9 0
Accumulated Reserve Account:		
Balance at 1.1.54	3,877 11 2	
Add Excess of Income over Expenditure	1,638 16 6	
		5,516 7 8
		16,233 19 8
Overdraft at Bank of New South Wales		3,776 13 5
		£20,010 13 1

We report that we have audited the accounts of the British Medical Association (Western Australian Branch) for the year ended December 31, 1954. In our opinion the accompanying Balance Sheet is properly drawn up and exhibits a true and correct view of the State of the Association's affairs as at 31st December, 1954, and the attached Income and Expenditure Account is also properly drawn up and exhibits a true and correct view of the Association's affairs for the year. Both are in accord with the best of the information and explanations given to us, and as shown by the Books of the Association.

(Sgd.) STOWE AND STOWE,
Auditors.

Perth, Western Australia, March 14, 1955.

Professor Gillies, Professor McWhirter and Sir James Learmonth from Edinburgh.

Professor Robson, Dr. Lendon and Dr. Poidevin also came here during Post-Graduate Week. Nearly forty practitioners of our own State have lectured or demonstrated.

Teams of lecturers have been sent to Bunbury, Narrogin, Kalgoorlie and Geraldton.

During Post-Graduate Week tape recordings were made for the first time of several lectures with the subsequent discussion. These have proved very popular with country groups.

Two all-day refresher courses were held, in midwifery and in ear, nose and throat. These were well attended, and similar one-day courses will be held in the coming year.

It had long been felt that a full systematic course of lectures, demonstrations and direction in anatomy and physiology could be held here. With the enthusiastic cooperation of a number of people this proved possible and a first class course suitable for the F.R.A.C.S. primary examination was held over a period of four months, and attended by over twenty doctors.

Six doctors received the sponsorship of the committee for overseas study. Much correspondence took place with the Director of Post-Graduate Studies in London concerning courses and accommodation, and a number of doctors were given advice and guidance.

Ship surgeon posts were obtained for three doctors.

Post-Graduate Week was designed to present a balance between high academic thought and generally useful practical advice. Professor Astwood and Professor Robson and Dr. Lendon and Dr. Poidevin with several of our own colleagues gave a varied programme which seemed to meet with general approval. You may recollect that over 300 doctors attended one of Professor Robson's lectures.

The monthly clinical meetings presented little difficulty in view of the number of visiting teachers. You may recollect the medico-legal evening and the talk by our friend the coroner. The medico-legal evening will be held again this year.

Further points from the committee's activities could be mentioned. Members have attended, at their own expense, meetings of the Post-Graduate Federation in Melbourne, and one member recently represented this committee at a meeting in Sydney to discuss the training of specialists.

Facilities for the training under the Colombo plan of Asian graduates are being investigated.

The committee carries out these commitments largely on a government grant. Plans are complete for the coming year and will be published soon. Some time, no doubt, this Medical Faculty will be largely responsible for post-graduate affairs. Until then this committee will continue in its duty.

British Medical Association House.

Dr. A. L. Dawkins, the Acting President, gave an account of the position regarding British Medical Association House in King's Park Road.

Medical Education.

THE TRAINING OF SPECIALISTS.

A CONFERENCE on medical education, dealing specially with "The Training of Specialists", was held at the Stawell Hall, 145 Macquarie Street, Sydney, on March 2, 1955. This was the second conference convened by the Post-Graduate Committee in Medicine in the University of Sydney and all members of the medical profession had been invited to attend. The first conference was held on May 3, 1954, and dealt with "Training for General Practice". The gathering took the form of a panel discussion. Dr. V. M. Coppleson, Honorary Director of the Post-Graduate Committee, occupied the chair. The members of the panel were Mr. Julian Orm Smith (Melbourne), Dr. T. M. Greenaway, Dr. S. Devenish Meares, Dr. A. R. Colwell and Dr. Selwyn Nelson.

DR. V. M. COPPLESON opened the conference and introduced the other members of the panel. He said that for many years specialist education in medicine had been confined to medicine and surgery, under the control of the Royal Colleges of London, England, Scotland and Ireland. Changes in recent years had included recognition of an increasing number of specialties, the institution of new diplomas, the establishment of Colleges in new specialties and the widening of the scope of the older Colleges, the adoption of a system of Specialty Boards in the United States of America, and more recently the institution of the registrar system of training in the United Kingdom, and the resident system in the United States of America. The most debated matters, apart from organization, had been to decide what subjects should be regarded as specialties to be covered by diplomas and specialist training, and to determine the method and length of training.

Dr. Coppleson expressed the hope that there would be discussion on the basic policy behind training and the granting of diplomas. He explained that in the United States of America diplomas were granted as an indication that the recipient was a highly experienced practitioner, mature in his specialty, whereas in the United Kingdom a diploma was an indication that the holder had undergone certain training and had satisfied examiners of his fitness to practise his specialty. The University of Sydney had followed the British system. However, Dr. Coppleson said, that did not mean that British communities had no method of recognizing superior merit; this was done by the granting of qualifications at various levels, so that while American universities had only one degree, British universities granted bachelor's degrees to recent graduates, master's degrees at teacher level, and doctor's degrees for higher awards; the Royal Colleges of Physicians granted membership by examination, but reserved their fellowship for more mature accomplishments, conferring them by invitation, not by examination. Dr. Coppleson's belief was that a more general acceptance of this principle of grading of qualifications would go far to rationalize specialist training and education. Outlining development in Australia, he mentioned the establishment of the Royal Australasian College of Surgeons, The Royal Australasian College of Physicians, and the Australasian College of Radiologists. He referred to the establishment in Australia of a Regional Council of the Royal College of Obstetricians and Gynæcologists. He stated that the Australian universities had interested themselves in specialist education much more actively than their counterparts in the United Kingdom, and instanced the diplomas granted by the University of Sydney, which were the same as those of the United Kingdom, except for the lack of a Diploma in Child Health; the Diploma in Dermatological Medicine was the only one in the British Empire, and though subject to some criticism, met local needs. Recently, on the advice of the Post-Graduate Committee, the University had raised standards and doubled periods of training, and the question of further extending some to three years was being examined. Investigation of the capacity of hospitals in New South Wales to train medical graduates had revealed that there were sufficient posts available for the specialties covered by the diplomas, but not for medicine and surgery. Dr. Coppleson emphasized that training in medicine and surgery in New South Wales was difficult to obtain. No system of registrar or resident training had been adopted by the teaching hospitals, while the associate system of training young surgeons advocated by the Royal Australasian College of Surgeons which had proved so successful in Melbourne had proved incompatible with the New South Wales hospital system. It was unfortunate that the University of Sydney had lost, since the war, its Post-Graduate Hospital, the Prince Henry Hospital. As a result, most young graduates from New South Wales were compelled to go abroad for adequate training in medicine and surgery. As a consequence, most new appointees to the teaching hospitals, especially in surgery, were holders of overseas rather than Australian qualifications, which was not a healthy situation, and required correction.

Dr. Coppleson explained that the Committee had found that candidates, several years after graduation, had difficulty in meeting the more advanced standards for diplomas, especially in physiology and biochemistry, and had considered the institution of two courses for Part I examinations, the first an optional revision course to bring the candidates up to diploma standard in subjects common to all diplomas, and the second devoted to the more special subjects.

The question had lately been raised whether the Australian universities should now cease issuing diplomas altogether, in favour of the Colleges, but there were difficulties, mainly geographical, which the speaker hoped the conference would discuss. The Committee had given its opinion that the diplomas in public health, clinical pathology, psychological medicine and tropical medicine and hygiene should remain with the universities, but that the others could be left to the Colleges, which already provided diplomas in all but dermatological medicine. The important

problems would concern organization among the Colleges and possibly decentralization.

DR. SELWYN NELSON described the situation in the United Kingdom in full detail. It was very complex, but the standard of specialist competence achieved was very high. In the United States of America the position was dominated by the requirements of 19 Boards which granted certificates of competency in 24 specialties. In addition to basic standard requirements, a period of three years of training was demanded, all or part of it under the residency system. There was also a fellowship system conducted by certain university graduate schools, leading to higher degrees, and many courses were supplied by graduate schools.

MR. JULIAN ORM SMITH first gave a description of the system of admission to Fellowship of the Royal Australasian College of Surgeons, as it had developed since the founding of the College. The two-year period of post-graduate training in posts approved by the College was, in Melbourne, catered for by the appointment of men holding the first part of a senior surgical qualification as associate assistant surgeons to senior surgeons, and of men with a senior surgical qualification as assistant surgeons. Mr. Smith conceded that while this arrangement was suited to conditions in Melbourne, it might not suit those of other cities. Meanwhile the University of Melbourne was finding the work of training for various diplomas very burdensome, but the College was unable to accept additional commitments.

DR. T. M. GREENAWAY pointed out that The Royal Australasian College of Physicians, considering that it could not well cope with any widespread system of specialist training, regarded as its first care the setting and maintaining of standards. Candidates came mainly from the registrar group, men who had served for some years in a teaching hospital or a university department, or general practitioners who had taken time off and returned to their teaching hospitals for a period of study. Dr. Greenaway's personal view was that the teaching hospitals afforded abundant training for specialists. However, if a well-organized training centre was needed, it might be argued that a post-graduate hospital would be necessary, and if this would provide up-to-date training in anatomy, physiology, biochemistry and pathology, he would be in favour of it.

DR. S. DEVENISH MEARES described the organization of the Royal College of Obstetricians and Gynæcologists. He pointed out that it was spread through the whole British Commonwealth, through its College Regional Councils. In Australia the College had organized teaching by overseas lecturers, and conducted resident and non-resident courses. Admission to membership was by examination of candidates who had occupied resident posts in general medicine and surgery and in obstetrics and gynæcology, with certain other qualifications.

DR. ALAN R. COLWELL explained that specialist training in radiology presented a difficult problem in Australia, particularly because of the many specialized sections now existing. He expressed his opinion that radiologists should be trained in registrar posts in the larger hospitals, and that at present there was insufficient uniformity in the qualifications and examinations throughout Australasia.

Dr. V. M. Coppleson then invited questions.

In answer to the question "Is the panel of the opinion that no matter what the specialty, a period of general practice is highly desirable?" DR. T. M. GREENAWAY stated that "with the advances in medicine, and the rapid absorption of facets of medicine from special advances in special clinics, a period in general practice, valuable though it is, is becoming impossible for many young men". Dr. Greenaway felt that five years' diversified service in a large general hospital would give experience comparable in some way at least to the very valuable "routine" work of the general practitioner. He did not think a period of general practice was essential to specialization; some candidates with no background of general practice had been very good. He hoped that those practitioners who were sufficiently keen to maintain their interest would be the nucleus for practising physicians and consultants.

When asked if The Royal Australasian College of Physicians would be prepared to sponsor a dermatology diploma similar to the diploma in dermatological medicine, Dr. Greenaway said that the matter was under discussion, but was linked up with the question of whether the University or the Colleges should manage the diplomas, and he would prefer the question to be referred to the President of The Royal Australasian College of Physicians.

DR. E. W. FRECKER asked what was the attitude of the panel to the registration of specialists, and within what time limits?

MR. ORM SMITH stated that Fellowship of the Royal Australasian College of Surgeons was registerable in Victoria and in the United Kingdom at least, without regard to whether it had been granted in general surgery or specialty.

SIR HUGH POATE explained that specialists were registered in Queensland and Western Australia, but the other States had not adopted this. His opinion was that it would probably afford better protection for the general public if some efficient scheme could be devised for the registration of specialists in all branches of medicine.

DR. COTTER HARVEY pointed out that the matter was receiving attention from Medical Boards in all States, but so far no generally acceptable formula had been evolved.

DR. HAROLD HAM asked what steps the panel suggested should be taken to provide more hospital appointments for trained junior specialists.

MR. ORM SMITH replied that under the associate system a senior surgical qualification was required, and there were 20 such positions in general surgery available in Melbourne, of which 18 were occupied, and that to create too many would be unwieldy and expensive.

DR. S. DEVENISH MEARES explained that there were 27 posts available for men wishing to train for the membership of the Royal College of Obstetricians and Gynæcologists.

DR. SELWYN NELSON pointed out that there was a dual need for training specialists and general practitioners, and the creation of training posts for specialists would restrict the posts available for general practitioners. If additional posts were needed, there would have to be established a graduate training hospital in addition to the undergraduate training hospitals.

PROFESSOR B. T. MAYES opened the general discussion, speaking on general terms and not as a representative of any college. He referred to the necessity of spending three to five years in an approved hospital in training as a specialist. In the United Kingdom, however, in gynæcology there were far more hospital-trained specialists than the country could absorb, and many were forced back into general practice, where they were not at all satisfactory owing to their long lack of any contact with general medicine. In Australia it would be better to suit the training to local conditions. At least part of preliminary training should be done in Australia, even if the rest was done abroad. Professor Mayes preferred the specialist diploma as a certificate of competency rather than a badge of the elect. On the question of the granting of diplomas, the universities were the only places with practical facilities for teaching Part I subjects. With regard to general practice and specialization, Professor Mayes said that he had been disappointed in Dr. Greenaway's statement, and could not agree with him completely when he said that it was difficult for young men who had spent five years in hospital learning a specialty. It was sometimes hard on them, going straight from hospital to Macquarie Street; a lot of them went into general practice, which provided a good deal of obstetrics, and general practice was advantageous in more than one way. Professor Mayes thought that there might be a little too much institutional training and not enough contact with humanity in producing the specialist today.

SIR HERBERT SCHLINK reminded the Conference that the discussion concerned conditions as they existed in Australia, not in the outside world. The Colleges and the Post-Graduate Committee deserved every credit for the groundwork they had done. Sir Herbert Schlink stated that the universities should be the only examining bodies for the diplomas, as they were for the degrees. Their unwillingness to continue with the diplomas was entirely due to lack of funds and trained personnel; in New South Wales such lack was because, with only three and a half million people, the State was trying to run three universities with insufficient funds. His second point was that the Colleges were only specialist clubs, and the only examinations they should conduct were those for the admission of members. Thirdly, the teaching hospitals were the most important centres of specialist training and activity. A post-graduate hospital would be eventually desirable, but was financially impossible now. The teaching hospitals could take more apprentices for higher qualifications if they had the necessary finance. There was, fourthly, the necessity for strong post-graduate organizations to coordinate and cement the activities of the universities, teaching hospitals, specialist colleges, and the further education of the general practitioner.

DR. DOUGLAS ANDERSON stated that no person was permitted to practise until he had a certain educational background, a certain professional training, and a certain basic experience; in the case of general practitioners, the first two were the province of the universities, and the last of the

teaching hospitals. If certain categories of doctors were to be recognized, particularly by law, as specialists, then these three basic requirements should apply, and should be properly defined. The educational background could perhaps be established by a common primary or preliminary examination for all candidates seeking training as specialists. Mr. Orm Smith had shown how specialist training was given individually under the associate system. Apart from such special cases, the training of specialists demanded hospital posts and beds and out-patients in sufficient numbers, which meant strengthening the teaching hospitals. Specialist training centres within the larger hospitals were the best solution—separate centres were not desirable.

DR. J. C. BELISARIO stated that English dermatologists considered that the diploma in dermatological medicine should be abandoned in favour of the membership of The Royal Australasian College of Physicians. American dermatologists favoured the Australian system. It was not possible to obtain both qualifications without too heavy an expense. The diploma in dermatological medicine was virtually a double diploma already, in that it included such subjects as X-ray and radium therapy as well. It was impossible to keep up with general medicine and dermatology at the same time. There was not adequate hospital training in dermatology anywhere in the country.

DR. GORDON HISLOP wished to correct the statement that Western Australia had registration of specialists; the scheme had been provided for, but owing to its unpopularity with all sections, it was withdrawn.

DR. NORMAN WYNDHAM agreed with Mr. Orm Smith about the need for training surgeons, but suggested that conditions were not so bad in Sydney as Mr. Orm Smith had hinted. Fellowships in surgery had been in existence in Sydney, and in 1954 one was advertised and there was no applicant. Full-time registrarships in surgery were available at two hospitals in Sydney.

DR. J. J. SMYTH pointed out discrepancies between what had been regarded as desirable in surgical training by the Royal Australasian College of Surgeons, what was actually required by the College, what was asked of the hospitals by the College, and what was recommended as necessary for an appointment as a consultant for the National Health Services in the United Kingdom by the Royal College of Surgeons.

DR. GEORGE HALLIDAY pointed out that owing to war service many men had had to take diplomas rather than spend the time on qualifying for a fellowship. Latterly, however, registrarships at one Sydney hospital had been available which provided good specialist training, and a few more were desirable.

DR. V. M. COPPLESON then thanked the Colleges for sending such able representatives. He expressed the appreciation which the Post-Graduate Committee felt for the great help it had had from the English Post-Graduate Federation. He then declared the conference closed.

Medical Societies.

PÆDIATRIC SOCIETY OF VICTORIA.

A MEETING of the Pædiatric Society of Victoria was held on March 9, 1955, at the Royal Children's Hospital, Carlton, Victoria.

The Diagnosis of Hæmorrhagic Disorders.

DR. J. COLEBATCH introduced a discussion of the diagnosis of hæmorrhagic disorders by saying that the object was to present some of the results of the rapid growth of knowledge concerning the coagulation of blood. In all ages back to antiquity, bleeders had been recognized. The Greeks described what appeared to have been scurvy, under the name "stomakake". Severe familial bleeding, probably hæmophilia, was referred to in the Jewish Talmud, and again in the eleventh century by the Spanish-Arabian physician Albucasis. Two hundred and twenty years before the present day, *purpura hæmorrhagica* was described as an entity by Paul Werlhof, distinguished physician and poet of Hanover. By the beginning of the nineteenth century, comprehensive accounts of scurvy by Lind and of hæmophilia by Otto had further established those three classical bleeding diseases as separate entities. In the next hundred years Schönlein, Henoch and Osler, from the large pool of unidentified purpuras, drew a clear picture of so-called allergic purpura as another entity. Donne, who worked at the Paris Children's Hospital, discovered the platelets, and in the 1880's the work of Hayem and of Krause pointed to thrombocytopenia as the cause of bleeding in Werlhof's disease. Ehrlich's pioneering work on cell microchemistry and the rapid development in the field of physiology of what Castiglioni called neo-humoralism had, by the turn of the century, led to the classical theory of blood coagulation.

At that time, in 1905, it was known from the studies of Schmidt, Hammarstein and Morawitz that the insoluble fibrin of blood clot was formed from a soluble precursor, fibrinogen, through the action of thrombin, which was an albumin-like protein. Thrombin was not normally present in the blood, but it was thought to be derived from an inactive precursor, and that postulate was proved correct when the substance was isolated by Nolf in 1928 and found to be a globulin, which was later called prothrombin. Calcium was recognized as essential in the early stages of coagulation. Tissue extracts were known to induce clotting *in vitro*, but there was uncertainty about the natural source of the factor that initiated clotting *in vivo*. Morawitz postulated that the platelets supplied this factor and that it was an enzyme, called thrombokinase by him and subsequently renamed thromboplastin. He was substantially correct, as later workers had shown. A further step of clinical importance was taken in 1912, when George Whipple, then a morbid anatomist, postulated prothrombin deficiency as the cause of hæmorrhagic disease of the newborn. Two decades later the work of Kugelmass, a pædiatrician, provided support for that hypothesis, and in 1939 the story was rounded off when Dam and other workers independently showed that vitamin *K* could prevent and cure the hypoprothrombinæmia. The soundness of the fifty-year-old classical theory of blood coagulation propounded by Morawitz had been abundantly demonstrated, particularly by the fact that it formed the basis of all the investigation techniques in use at the present time. The enormous amount of recent research on coagulation factors had, however, revealed inadequacies in this classical theory. The modifications made necessary by modern knowledge of the thromboplastin complex and of the prothrombin complex were then discussed.

Dr. Colebatch proceeded to use this deliberately brief account of the coagulation of the blood as the basis for a discussion of the clinician's approach to the diagnosis of hæmorrhagic disorders. This part of his presentation will shortly be published in full as a separate paper. It dealt with the significance of a good history, including the family history, with the importance of a full clinical examination, with the use of the capillary fragility or tourniquet test, and with the information which the practitioner accustomed to doing his own clinical pathological work might obtain from simple tests such as the blood count and film examination, determination of the bleeding and clotting times, and the study of clot retraction. Finally, the application of the clinical and laboratory findings to the diagnosis of the more common hæmorrhagic disorders was presented, illustrated by a table.

In conclusion, Dr. Colebatch said that from his remarks, and from Miss Wilson's to follow, he hoped that it would be apparent that the diagnosis of hæmorrhagic disorders might not be simple, but that it could be tackled in an orderly fashion, that it must commence at the clinical level, and that laboratory aid was frequently indispensable.

Laboratory Aspects.

MISS BETTY WILSON stated that from Dr. Colebatch's short historical review it could be seen how rapidly the theories of blood coagulation and the discovery of new factors had progressed. The classical theory of Morowitz, which was proposed in 1905, intimated that prothrombin was a single substance which was quantitatively converted to thrombin under the influence of thrombokinase and calcium. Fibrinogen was converted to fibrin in the presence of thrombin, and the speed of the reaction was controlled by the amount of thrombin present. Of the factors involved in this reaction, prothrombin was a hypothetical substance, which could not be generally accepted at the time it was proposed. Nevertheless, the hypothesis of the existence of prothrombin led to much experimental work, and in 1914 prothrombin was isolated from plasma and its properties were studied. Quantitative methods for the measurement of prothrombin were then made possible. They assumed importance because of their clinical value in the assessment of liver disease. As more experimental work and observations were made, it soon became apparent that the classical theory could not explain all the phenomena of blood coagulation. Although prothrombin was the one essential precursor of thrombin in whole blood, various accelerating substances which determined the speed of the reaction were necessary for effective

thrombin formation. Thus prothrombin could be regarded, not as a single substance, but as a group of factors all required for thrombin formation. It was known that blood clotted readily when withdrawn from the body or when it was extravasated into damaged tissues, but in the vessels it remained fluid. In the living body coagulation was hindered by delay in the formation of activating factors. Also neutralizing factors such as antithrombin were present. An excess of one of those substances could be the cause of a hæmorrhage. The thrombin-fibrinogen reaction had been studied extensively and was generally accepted. As to the sequence of events in the early phases of coagulation, many theories existed but none was generally accepted. One stated that the thromboplastin complex existed in the blood as precursors, which became activated in contact with torn blood vessels or glass. The amount of thromboplastin so formed was only sufficient to initiate clotting; but in the presence of adequate numbers of platelets and calcium, a maximum formation of thromboplastin took place, and complete conversion of prothrombin into thrombin occurred. Accelerators and initiators influenced the rate of thrombin formation.

Because the equation for the coagulation of shed blood could not be expressed by precise chemical formulæ, many workers in that field had contributed a bewildering array of names for the various components necessary in the coagulation of blood. Miss Wilson then gave the nomenclature used by the Children's Hospital laboratory and the synonyms found in the literature. They were as follows:

Disease.	*Factor Deficient.*	*Synonym.*
Hæmophilia.	Anti-hæmophilic factor.	α Prothromboplastin.
Christmas disease.	Christmas factor.	Plasma thromboplastin component. β Prothromboplastin.
PTA deficiency. Hæmophilioid state D.	Plasma thromboplastin antecedent.	
PTF/D deficiency.	Plasma thromboplastin factor/D.	
Prothrombin deficiency.	Prothrombin.	Thrombogen.
Factor V deficiency. Hæmophilioid state A. Parahæmophilia.	Factor V.	AC globulin, labile factor, proaccelerin.
Factor VII deficiency. Hæmophilioid state B.	Factor VII.	S.P.C.A. proconvertin stable factor.

Miss Wilson said that fortunately synonyms for calcium and fibrinogen appeared to have been overlooked. A lack of any one of those factors or an excess of an inhibitory substance would result in a hæmorrhagic diathesis. A prerequisite for the understanding of the laboratory differentiation of the thromboplastin and related factor deficiencies was a knowledge of the more important properties of the factors, for it was on those properties that laboratory tests were based.

The thromboplastin complex contained at least three factors, antihæmophilic factor, Christmas factor and plasma thromboplastin antecedent. Anti-hæmophilic factor (A.H.F.) was deficient or present only in small amounts in the blood of hæmophiliacs. It was present in normal plasma and in the plasma of patients with any of the other coagulation factor deficiencies. A.H.F. was a globulin and found in the 0·33% saturated ammonium sulphate fraction of normal plasma. A.H.F. was consumed during clotting, and therefore it was not present in serum. It was not absorbed by chemicals such as barium sulphate. When normal plasma was stored, its A.H.F. content diminished, although its stability was enhanced at low temperatures. Although various workers had reported the correction of clotting times on the admixture of bloods from apparently classical hæmophiliacs, it was not until 1952 that another thromboplastin component (Christmas factor) was isolated and the characteristics that differentiated it from A.H.F. factor were demonstrated. This factor was missing from the plasma of patients with a hæmophilioid condition known as Christmas disease. It was present in normal plasma and in the plasma of persons who lacked any of the other factors. It was a globulin and present in the 40% to 50% saturated ammonium sulphate fraction. It was not consumed during clotting, and unlike A.H.F. was present in serum. The Christmas factor was absorbed by barium sulphate and removed from normal plasma by Seitz filtration. It was stable in plasma, blood or serum stored at room or refrigerator temperature.

Plasma thromboplastin antecedent (P.T.A.) was another thromboplastin component identified in 1953. It was present in normal plasma and serum and also found in adequate amounts in plasma from patients with either hæmophilia or Christmas disease. P.T.A. was found in the 20% to 30% saturated ammonium sulphate fraction of normal plasma. It was thought not to be absorbed by barium sulphate, but the eluate of absorbed normal plasma would correct a P.T.A. deficiency. A fourth factor was discovered in January, 1954, and named plasma thromboplastin deficiency/D. One patient with a bleeding disorder due to the absence of this factor had been recorded in the literature.

Miss Wilson said that prothrombin was produced in the liver and found in normal plasma. It could be quantitatively converted to thrombin in the presence of factor V, factor VII, thromboplastin and calcium. Factor V, isolated in 1944 from normal plasma, was labile and not absorbed by barium sulphate. Fourteen cases of bleeding disorders due to a deficiency of this factor had been recorded. Factor VII was isolated in 1951. It was present as a precursor in plasma, but was not easily separated from prothrombin. Factor VII was stable and absorbed together with prothrombin by barium sulphate. Fibrinogen was produced in the liver and might be defined as the plasma protein clotted by thrombin. A deficiency of fibrinogen was a rare cause of defective blood coagulation; but if the plasma content of fibrinogen dropped below 60 milligrammes per 100 millilitres, a hæmorrhagic diathesis could result. Afibrinogenæmia could be a congenital deficiency or could be acquired in conditions associated with impaired liver function.

If a patient was referred to the laboratory with a history of hæmarthroses, recurrent bruising, bleeding from minor trauma *et cetera*, the clinician might suspect that some disorder akin to hæmophilia was present, but in order to exclude any other possible blood disorders a full examination was made of peripheral blood including estimation of the bleeding time and platelet count. The normality of the findings, other than perhaps a hæmoglobin value lowered as a result of hæmorrhage, was the signal for the investigation of the two complexes mentioned previously, the thromboplastin complex and the prothrombin complex. The clotting time of whole blood was first estimated by Lee and White's method and confirmed by the more convenient recalcified clotting time method. The latter test was more convenient since it could be performed under standard conditions and when convenient to the laboratory staff. Venous blood was used for the examination of coagulation times and all the subsequent tests. Blood was taken by means of syringes lined with silicone into similarly prepared tubes. As contact of blood with glass would initiate the formation of plasma tromboplastin, the use of an agent such as silicone was necessary to delay this early phase and allow minor deficiencies to be detected. Adequate amounts of prothrombin were present in the blood of subjects with thromboplastin deficiencies; therefore the result of the ordinary one-stage prothrombin estimation would categorize the patient's defect into the thromboplastin complex or the prothrombin group. If the prothrombin value was low, a lack of factor V, factor VII or prothrombin was suspected. Further tests were done to differentiate lack of factor V and factor VII from a pure prothrombin deficiency. It was perhaps of interest to note at the present point that plasma of patients being treated with the dicoumarin group of drugs lacked factor VII. Experience in the laboratory showed true prothrombin deficiencies to be limited to liver disorders, and therefore determinations of the prothrombin level appeared to be a reliable function test. Diseases of the newborn had not been investigated, nor had those of any patient in whom absence of factor V or VII was the cause of bleeding. Had the prothrombin been found normal, then the defect would appear to be due to the lack of a factor in the thromboplastin group, an inhibitor or perhaps an anticoagulant, and investigations proceeded in the following way. During coagulation of normal plasma most of the prothrombin was converted to thrombin, so that very little was found in the serum. Thus subtracting the level of prothrombin in serum from that of plasma enabled an estimate to be made of the amount of prothrombin consumed. In the coagulation of blood which lacked a thromboplastin factor, very little prothrombin was converted to thrombin—in other words, the prothrombin consumption was low. At that stage an effort was made to pinpoint the specific factor, the lack of which was responsible for the disorder of coagulation. Once again, use was made of the recalcified clotting times. On the addition of normal plasma to the patient's plasma, the clotting time should be corrected. If the time taken for the plasma to clot was prolonged, an inhibitor or circulating anticoagulant could be the cause of bleeding. Additional tests were carried out to determine the specific substances involved. The time taken for the patient's plasma to clot

was known, and then varying amounts of plasma naturally deficient in one of the few components—for example, plasma from a known hæmophiliac, or plasma made deficient by barium sulphate—were added.

The addition of plasma or serum containing the missing factor would correct the clotting time of the patient's plasma. On the addition of plasma or serum in which the missing factor was not present the clotting time would remain unaltered. For the use of such methods, reserves of plasma from patients with known factor deficiencies were necessary. As those were not always available at the Royal Children's Hospital, another and perhaps better approach to the elucidation of the missing factor could be made by means of a test known as the thromboplastin generation test. This test allowed each factor necessary for coagulation to be assayed separately, and it was possible to identify and measure the deficiency, without relying on an array of stored plasma samples. Until recently the test was time-consuming and complicated, but recent modifications had made it practicable in routine work. Whilst the hæmorrhagic disorders were being investigated in a routine laboratory, one came up against difficulties and equivocal results for which no satisfactory answer could be given. That might, of course, lead to the isolation of more new factors. However, with the discovery of even more new factors, the investigations had not been simplified but had been made more complex.

Miss Wilson concluded by saying that she thought the greatest difficulty experienced by the laboratory staff at a pædiatric hospital was in attempting to perform the maximum number of tests with the minimum amount of blood.

DR. J. WILLIAMS asked Dr. Colebatch if it was known whether the emotional state of the patient influenced the coagulation time of the blood. He said that Cannon had stated in his book on "Fear, Hunger and Pain" that there was a decreased bleeding time in those states.

Dr. Colebatch replied that he did not know of such an effect.

DR. R. SAWERS said that it was known that an acute fibrinolysin might develop in the blood in such states.

DR. KATE CAMPBELL said that she had recently tried to stop bleeding from the circumcision wound in a hæmophiliac by the application of a pad soaked in breast milk. That had worked successfully.

Dr. Colebatch replied that breast milk was known to contain a significant amount of thromboplastin (the anti-hæmophilia factor).

DR. J. MCLEAN raised the question of the specificity of hæmarthroses in hæmophiliacs. He had had a patient of sixty-eight years with a hæmarthrosis but no previous history of bleeding. When the patient's blood was added to normal blood, it prevented the normal blood from clotting. The blood contained an anticoagulation factor.

Dr. Colebatch, in reply, said that anticoagulation factors were recognized but constituted a very rare problem in children. Hæmarthroses were almost specific to hæmophilia.

DR. A. WILLIAMS, in supporting Miss Wilson's paper, said that in such a short discussion as the present one many aspects of bleeding, such as those of circulating anticoagulation factors, abnormal platelets and absent fibrinogen, had had to be omitted from the discussion. He said also that many atypical cases of bleeding tendencies were always occurring, and that was one of the reasons for continuing with the investigations. Some might ask if it was worth investigating the cases so thoroughly. He thought that the answer was definitely "yes". At least the distinction between Christmas disease and hæmophilia had shown that stored blood was the correct treatment for patients with the first and fresh plasma for those with the second. However, he thought that there was no need to investigate the siblings of subjects of hæmophilia as newborns; that could wait until the second year.

DR. J. MCLEAN asked what was the method of obtaining blood from the infants, and if the internal jugular vein was ever used.

DR. V. COLLINS answered the question and said that the external jugular or the superficial arm veins were usually used. The femoral vein should not be used.

DR. M. HUTSON asked whether there was any clinical difference between Christmas disease and hæmophilia, and whether the age of onset differed, or whether there was a difference with age in the tendency to hæmorrhage.

Dr. Colebatch, in reply, said that the content of anti-hæmophilia factor in the blood of adults did not vary, although in children the tendency to hæmorrhage seemed to vary. That might be due to secondary factors such as infections and vascular damage. Christmas disease and hæmophilia were clinically identical, and both might become manifest at circumcision.

Dr. V. Collins asked Dr. Colebatch what was the latest view of the ætiology of hæmorrhagic disease of the newborn.

Dr. Colebatch said that he and Miss Wilson had not studied newborn hæmorrhage in detail, but the Scandinavian viewpoint now was that in the full-term baby the condition was due to a deficiency of the prothrombin complex. In premature babies, however, there was the added factor of cell fragility, and the prothrombin level might not be related to the degree of hæmorrhage.

Genetics of Hæmophilia.

DR. R. SAWERS, from the Baker Medical Research Institute, Alfred Hospital, presented a paper on the genetics of hæmophilia. He said that his comments would be of a rather dogmatic nature, firstly, to present a clear picture and, secondly, he hoped, to provoke discussion. He referred to hæmophilia as a syndrome and said that the two diseases belonging to the syndrome were α hæmophilia and β hæmophilia, the plasma factor deficient in each being α and β prothromboplastin respectively.

The genes responsible for the normal production of those substances resided in that part of the X chromosome having no homologous part in the Y chromosome. As far as was known, the genes were completely independent of one another and would be referred to in the talk as A and B respectively. The abnormal alleles were referred to as a and β respectively. Dr. Sawers said that he believed the genes and a and β to be incompletely recessive. He then showed lantern slides to demonstrate the genetic patterns for males and females, and the mode of inheritance. Normal women having two X chromosomes had the genotype AB/AB, and the male with one chromosome had the genotype AB/−−. A female carrier would on an average have one carrier daughter, one hæmophiliac son and a normal son and normal daughter. Of the children of a male hæmophiliac all daughters would be carriers of the disease and all sons would be unaffected.

At this stage Dr. Sawers mentioned the fact that the genes responsible for colour vision were carried on the X chromosome. The easily detected abnormal allelomorphs were carried by about 10% of people. In his series there were two pedigrees in which hæmophilia and colour blindness were associated. These might be of assistance in genetic and eugenic studies.

Dr. Sawers said that he and Dr. Fantl at the Baker Medical Research Institute had to date examined 70 hæmophiliacs and knew of at least 30 more. That gave an incidence of about 85 per million males, which was double the Danish figures of Andreassen. The birth incidence was one in 5000 males, and the average age at death was between twenty and thirty years—in other words, one-third to one-half the normal life expectancy. From those figures the number of living persons affected in Victoria would be expected to be in the region of 100. The figures were approximate, as the bulk of the persons affected had been contacted through the metropolitan public hospitals only.

In discussing hæmophilia, Dr. Sawers said that it was due to a series of allelomorphs. The abnormal allele, a, consisted of a continuous series of alleles which differed in degree of deviation from the normal A gene. It was convenient to divide the series into four groups: a' (0% prothromboplastin), a'' (trace to 5% prothromboplastin), a''' (6% to 10% prothromboplastin), a'''' (11% to 25% prothromboplastin).

The inheritance of the degree of defect in a pedigree was constant. To date, eight pedigrees of β hæmophiliacs had been investigated, and only complete deficiency had been shown. That was also the commonest type in a hæmophilia.

It was generally acknowledged that the majority of hæmophiliacs died without reproducing, and thus there was a continuous loss of abnormal genes. The disease would rapidly die out were it not for the occurrence of mutations (a sudden change of a normal gene to an abnormal one). Thus cases would be expected to occur in pedigrees with no previous family history of the disease, and in fact 34 of their 70 subjects had no previous family history. Also those pedigrees with the severe abnormality of A had the highest

occurrence of "new cases" (cases due to recent mutation). Those unexpectedly high figures had caused some concern until calculations were made. When the reproductive fitness was low—in other words, in severe cases—the proportion of affected persons without previous history was high, and the reverse was the case when the disease was mild. For hæmophilia as a whole, reproductive fitness was approximately 0·3%, and the expected figure for subjects without family history was 40% to 50%, whilst the observed figure was 48%.

Mutation rates were of increasing importance in the present atomic age. A rough estimate of the mean mutation rate per gene for the hæmophilia syndrome was $5{\cdot}8 \times 10^{-5}$, approximately double that calculated by Haldane.

Dr. Sawers then went on to discuss eugenic counselling. He said that in families with a long history of the disease, the risk that a woman would bear hæmophiliac children could be calculated, and he hoped that later by coagulation studies risks could be determined absolutely. A problem arose with female relatives of "first cases", and one did not know where the mutation had occurred in the pedigree. He then discussed an interesting example of this. A female cousin of a hæmophiliac child had desired to know if she might be expected to be a carrier. Review of the pedigree suggested that that was unlikely. Her child was, however, a hæmophiliac and on testing proved to have β hæmophilia. The individual deficiencies were corrected when the child's blood was mixed with that of his hæmophiliac cousin. The grandmother might then have been expected to have the genotype $\alpha B/A\beta$, in which case all her daughters would be carriers. That appeared unlikely, as two other daughters had six normal male children. The grandmother could have been $\alpha\beta/AB$; then the two daughters with hæmophiliac descendants would have genotype $\alpha\beta/AB$. One would then have to suppose a cross over in each chromosome in the gametes during their formation. No data for cross-over rate at that site had been computed, but the chance was probably of a very low order. The most likely explanation was the occurrence of two independent mutations.

In concluding, Dr. Sawers said that the genetic study that he and Dr. Fantl had made had been of special value, as biochemical characterization and quantitative evaluation had been performed in each case studied. In Victoria there was a relatively fixed population, and statistical data could be obtained from birth rates *et cetera*. Further, the size of the population was satisfactory for obtaining enough cases, yet not too large for laboratory study. However, the human species was a particularly difficult one for genetic study. It appeared that increasing importance was being attached to mutations in modern atomic civilization, and their present study could be the basis for comparative studies later. To date they had tackled one source of cases only, the metropolitan hospitals. He would be most grateful to hear of new hæmophiliac families.

Dr. Sawers said that he was grateful to all the hospitals who had helped with the supply of case material, and to the Department of Statistics at the University for help in analysing the data.

DR. G. WEIGALL asked Dr. Sawers whether the association of hæmophilia and colour blindness was only that they were both transmitted in genetic fashion or whether there was any other connexion.

Dr. Sawers said that they were independent, but if associated in a pedigree might by "cross over" become dissociated.

DR. A. WILLIAMS asked what was the ratio of cases of Christmas disease to those of hæmophilia. He asked whether the inheritance of hæmophilia was incompletely recessive and whether there were other diseases inherited in an incompletely recessive manner.

Dr. Sawers replied that in a series of 70 cases there were 10 cases of Christmas disease. Regarding Dr. Williams's second question he said that he had a strong impression from the history of mothers of children with hæmophilia that they were frequently mild "bleeders" themselves. Dr. Fantl had described a woman who had mild hæmophilia, while her son had the complete deficiency disease. Brinkhouse, in one pedigree of mild hæmophilia, had found half the carrier mothers to be mildly deficient in the factor concerned. That was the reason for the earlier statement that the genes for hæmophilia were incompletely recessive. Similar findings had been reported in other hæmorrhagic diseases.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

FISH IN PORT JACKSON.[1]

[From "A Narrative of the Expedition to Botany Bay", by Watkin Tench, Captain of Marines.]

FISH which our sanguine hopes led us to expect in great quantities do not abound. In summer they are tolerably plentiful, but for some months past very few have been taken. Botany Bay exceeds in this respect Port Jackson. The French one day caught near two thousand fish in one day of a species of groper, to which, from the form of a bone in the head resembling a helmet we have given the name of light horsemen. To this may be added bass, mullet, skait, soles, leather-jackets and many other species, all so good in their kind, as to double our regret at their not being more numerous. Sharks of an enormous size are found here. One of these was caught by the people on board the Sirius which measured at the shoulders six feet and a half in circumference. His liver yielded twenty four gallons of oil, and in his stomach was found the head of a shark which had been thrown overboard from the same ship. The Indians probably from having felt the effects of their voracious fury, testify the utmost horror on seeing these terrible fish.

Correspondence.

CHILBLAINS.

SIR: The ætiology of chilblains remains obscure. When I was a young man with a vigorous circulation I suffered much from chilblains; now that I am old with a sluggish circulation I am never bothered with them though exposed to the same climatic conditions and taking no extra precautions for defence. In a recently published book, "Man in a Cold Environment", issued under the auspices of the Physiological Society of Britain, it is stated that this form of frostbite is "particularly common in adolescents". Now in youth the hands, feet and ears are warm; in age they are cold in wintry weather. I venture to think that the larger temperature interval between the extremities and the surrounding air may be a causative factor in the greater incidence in earlier years.

Yours, etc.,
W. A. OSBORNE.

Kangaroo Ground,
Victoria,
Undated.

THE RESERPINE ANTI-HYPERTENSIVE DRUGS.

SIR: Dr. Swanton (July 2, 1955) rightly draws attention to the unpleasant and occasionally dangerous side effects of the rauwolfia group of alkaloids. In carefully selected cases these symptoms are comparatively rarely met with, but, as Dr. Swanton suggests, he and his psychiatric colleagues see most of the patients with severe reactions and consequently may form an exaggerated opinion of the frequency of these disturbing complications.

Rauwolfia is a very valuable and potent addition to the therapy of hypertension, but like all potent drugs has to be used with care. Its great value is that the fall in pressure is gradual, the levels rarely fluctuate, and the diastolic pressure is always markedly reduced if the drug exerts a hypotensive effect.

It has been my experience that the drug is effective in only 5% of patients with severe progressive hypertension, but there is no way of telling which patients will react without trial. For this reason it is my practice to prescribe rauwolfia for three weeks to all patients who are com-

[1] From the original in the Mitchell Library, Sydney.

mencing treatment for hypertension. I am in entire agreement with Dr. Swanton that only patients with severe progressive hypertension with a raised diastolic pressure or those with complications resulting from a high blood pressure should be treated by any of the modern effective but occasionally dangerous drugs. I would not exclude patients with an anxious temperament, provided they have a high diastolic pressure, and the results of treating such patients are generally extremely satisfactory.

Dr. Swanton is absolutely correct in stating that the aged should not be treated for hypertension, nor should people of the anxiety depressive type whose systolic pressure alone is raised be subjected to therapy. These two groups are not in fact suffering from essential progressive hypertension. I further agree that patients being treated with rauwolfia should be under supervision, as the fall is often very great, being as much as 120 millimetres of mercury, systolic, and 40 millimetres of mercury, diastolic. The maintenance dose may only be one-third or one-quarter of the original dose, and consequently close observation is necessary. It has been my experience that if the patient is going to react to reserpine, the dose required rarely exceeds three-quarters to one milligramme. Increasing the dose seldom produces much further fall in blood pressure, but the incidence of reactions increases markedly. The maximum dose that I use, and that uncommonly, is 1·5 milligrammes. The toxic results are similar whether reserpine alone is used or the whole extract of rauwolfia. Most of the side effects are not as serious as those described by Dr. Swanton and consist of stuffiness in the nose usually relieved by an astringent spray, mild gastro-intestinal upsets, drowsiness and occasionally peculiar dreams. Some patients, as Dr. Swanton records, complain of intense physical fatigue and others of "horrible feelings" which they cannot define. The drug should be stopped in this latter group of patients. Marked increase of weight is not common.

I do not agree with Dr. Swanton that patients taking these alkaloids should necessarily be stabilized in bed, as it often takes weeks or months for a stable dosage to be estimated. It has further been my experience that the pressure falls slowly, and the toxic effects also develop gradually. Fluctuation in pressure and symptoms is very much less than with the ganglionic blocking agents. As yet there is no knowledge of the long-term effect of the drug on the liver or other organs. I have been using rauwolfia for three and a half years and have not yet noticed any new toxic effects developing with long use.

In conclusion, rauwolfia is a very useful and potent drug, severe toxic effects may occur occasionally, but with proper selection and care of the patients these effects should not be frequent and when present the drug should be stopped. Dr. Swanton has done a service in drawing attention to the fact that rauwolfia alkaloids should not be prescribed as freely as is happening at the moment. It is my impression that they are as casually prescribed as phenobarbital has been for years.

Yours, etc.,

55 Collins Street, Melbourne,
July 12, 1955.

J. G. HAYDEN.

SIR: Dr. Cedric Swanton's warning on the depressive effects of reserpine is timely, and the side effects of this interesting and expensive drug do require more ventilation than they receive in the trade literature. To produce any beneficial hypotensive effect, reserpine probably has to be administered in a dose of at least 0·5 milligramme daily, but one finds much larger amounts advocated and prescribed. Most patients taking the above dose complain of feeling "slowed down". This may be temporarily of advantage in the case of an aggressive or over-conscientious hypertensive, but if prolonged, the patient may become seriously depressed. As Dr. Swanton suggests, anticipation and more frequent observation will usually give the physician warning enough, but it should be remembered that some of the effects of the drug may continue for as long as two weeks after it has been omitted. Another subtle side effect is a tendency to increase of weight (fat as well as fluid), which may contradict attempts to reduce obesity in the management of hypertension. Unlike the barbiturates, there has not been in my experience any tendency to habit formation. In association particularly with digitalis, a reduction of intracardiac conduction time can occur, even complete heart block, in addition to the usual tachycardia induced by the drug. Other occasional side effects to be remembered are an increase in gastro-intestinal mobility, pupillary miosis, hypothermia, nightmares and agitation.

To give the drug at bedtime does not overcome excess torpidity or mental depression.

However, though there is little doubt that the drug is overprescribed in benign hypertension, it is a valuable agent in the treatment of more severe grades of hyperpiesia. Itself only weakly hypotensive, it seems to have the remarkable property of "holding" a depressed blood pressure level induced by other more potent agents, which can subsequently be lessened or even stopped altogether. Unlike the latter, reserpine appears to have no tendency to produce organic tissue damage after prolonged administration, and it would be a pity if we had to abandon its use because of its side effects. Dr. Swanton no doubt is correct when he suspects that psychiatrists see an increasing number of depressives whose illness is enhanced by the drug, and it is important that this situation should be appreciated; but there are many more recipients who do not suffer in this way, and where reserpine is of real help in blood pressure control.

Yours, etc.,

141 Macquarie Street, Sydney,
July 7, 1955.

KEMPSON MADDOX.

THE MEDICAL SERVICES OF THE AUSTRALIAN ARMY.

SIR: After ten years of peace and almost as many of "cold war" it is well to review the position of the medical services of the army. As a profession we have as always a very clear and definite duty in the provision of suitable, trained and devoted medical officers to take their place with the forefront of our nation's army. What has been our record over this vital ten years, during which those of us who were suitable in World War II have become "U.S."? It is one of which we should be heartily ashamed.

For a profession that is overfond of mouthing platitudes it is Gilbertian. From the highest to the humblest of us we have fallen down on our job, and today the army is lacking in a trained cadre of medical officers to organize the service in what will, if it comes, be the most stupendous task it will ever have to undertake.

When I was a young doctor it was the fashion of our honoraries to encourage by all manner of means, by word of encouragement, by reference during their operations, in discussion of medical cases, an interest in war medicine. I wonder do they do it today? Surely with the dreaful vista before us they should do all in their power to promote and encourage such an interest.

But no matter how many good doctors we have, unless we have the administratively and field trained officers all our efforts will be of no avail. No matter what a man's medical capabilities are they give him no "know how" to control men, to administer a unit, to follow the battle, to deploy his unit and so on. These things can only be learned the hard way—by serving in a medical unit. And these things are vital.

Unfortunately to learn these things in peace time means the using of valuable time, extra study, difficult and at times unpleasant disciplinary measures, and so on, all of which do absolutely nothing to further one's success in our chosen profession. Unless war does come, the profit and loss account will show largely losses. One must content oneself with the rewards of a conscience untroubled. Now I feel that this is where we have fallen down on our job—the demands of service come on the young men, and the older ones get it both ways. They smugly sit back and rest on a record of service in a bygone war, fondly imagining that should be enough and it is up to the young fellow to carry the burden while they enjoy the ease, leisure and fullness of a well-rounded life. That attitude is to be deprecated in full measure. To paraphrase Montgomery, "as long as he has breath in his body" any medical officer's service should be at the command of the country. If these senior men would only advise, encourage and assist these younger men, we would not be in the sad plight we are today—medical units under strength in medical officers.

Surely it should not be too much for the Colleges to insist on a confirmation of a first appointment to be a prerequisite of entry. It is a fact that the Corps Mess is dead—cannot it be resurrected into a vigorous organization? How many of us wear the Corps tie and are proud to do so? Who thinks to address our senior officers by their military title? Surely a brigadier or a full colonel should always be accorded his title, even in a teaching hospital.

By many and manifest ways an interest and, I hope, a fondness for the army can be developed, but let it be given to our younger fellows as cheaply and as pleasantly as possible, for it is their shoulders and not ours that will bear the burden.

Yours, etc.,

THOMAS J. RITCHIE.

55 Oxford Street,
Bondi Junction,
New South Wales.
July 11, 1955.

THE CHRISTIAN MEDICAL FELLOWSHIP OF AUSTRALIA.

SIR: Following the tradition of the last session of the Australasian Medical Congress, and of similar congresses in the United Kingdom and in Canada, the Christian Medical Fellowship of Australia has arranged a meeting to be held at the Stawell Hall, 145 Macquarie Street, Sydney, on Sunday, August 21, at 2.45 p.m. Dr. H. H. Willis, New South Wales State President of the British Medical Association, has kindly consented to take the chair. Dr. James Isbister, Dr. Ronald R. Winton and Dr. J. Gavin Johnson will speak on "The Relevance of Christianity to Medicine". All interested members of the profession and their wives are cordially invited to the meeting and afterwards to afternoon tea at the home of Dr. and Mrs. John Hercus, 1 Clark Road, North Sydney.

Yours, etc.,

DOUGLAS TRELOAR,
New South Wales Secretary for the Christian Medical Fellowship.

135 Macquarie Street,
Sydney,
July 18, 1955.

CONVENIENT BACTERIOLOGICAL SERVICES.

SIR: The interesting little booklet "Chemotherapy with Antibiotics and Allied Drugs", just distributed by the National Health and Medical Research Council, makes fascinating reading indeed. To translate it into practice, the building up of convenient bacteriological services seems highly desirable. Such services are provided elsewhere. I mean: (a) free supply of sterilized tubes, tubes with swab-wires and stoolspoons for all medical practitioners; (b) free provision of wooden blocks for safe transmission by mail; (c) provision of mail-free, strikingly coloured envelopes with printed address of regional bacteriological facility, marked "Urgent", "Infectious Material" *et cetera*, with a general directive to "P.M.G." personnel to give these letters high priority; (d) speedy communication of result of bacteriological and serological tests. Culture methods are still much slower than is ideal for general practice, and any delay in treatment costs most probably loss of working days. It would hence be very helpful if results would be communicated or at least reach the mail the same day as they become known to the bacteriologist.

Yours, etc.,

HANNS PACY.

Tea Gardens,
New South Wales,
July 17, 1955.

THE SALK VACCINE AND A PRECAUTIONARY SUGGESTION.

SIR: Despite reassurances, and in the presence of far from completed revised controls, the use of Salk vaccine still offers its vast values with imperfections which cannot but occasion anxiety in the minds of parents and recipients.

In the United States of America reports based on laboratorial, governmental and medical congressional announcements, we are informed: (i) that three firms have produced live virus-containing vaccine, one being responsible for paralytic sequels far in excess of what is to be expected of the sporadic incidence of poliomyelitis; (ii) that, in a combined national committee, assembled to finally adjudicate on immediate or delayed precautionary use of the vaccine, proponents *versus* antagonists were ranged in a proportion of seven to three respectively; while Dr. Salk preferred not to contribute to the verdict, leaving the decision to others. While paying tribute to the great national service rendered by Dr. Bazeley *et alii* in carrying out the specialized spade work in making this agent available to Australia, my point in writing is to diffidently tender a technological suggestion, which may obviate long-range precautionary experimentation before a situation of complete and safe reliance is assured.

What appears to be the most significant objective conclusion arrived at in the United States of America, as to the reason for retention of a minute residue of live virus in the commercial vaccine, is that in mass production a clumping of dead virus may encase an occasional live virus in a membrane impermeable to formaldehyde.

In a wide sphere of immunology and ecology, I have had some experience in the positive value of spreading factor, hyaluronidase, which as a secretion of synovial membrane possesses physiological percolating function. This is probably applicable in a different context to the in-vivo vaccine produced in living cultural kidney tissue. Spreading factor also causes active dispersion of chemical and metabolic agents, and may condition formaldehyde on a total homogeneous basis. The initial tests could be done on monkeys by adding spreading factor to suspect vaccine.

Yours, etc.,

FRANK TRINCA.

111 Collins Street,
Melbourne,
June 25, 1955.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE following notes relate to the Ninth Session of the Australasian Medical Congress (British Medical Association) to be held at the University of Sydney from August 20 to 27, 1955.

Executive Officers.

The Executive Committee records with deep regret the death of Sir Archibald Collins. Sir Charles Bickerton Blackburn has accepted the invitation to preside at Congress, and all members are indebted to him for consenting to undertake the onerous responsibility at short notice.

Lady Collins has expressed the desire to continue to preside over the Ladies' Committee, and this offer has been accepted with gratitude.

Locations of Principal Activities.

The principal activities will be located as follows:

Registration: The Union Hall, University of Sydney.

Social Club: Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street.

Official Opening: 8.30 p.m., Monday, August 22, Sydney Town Hall.

President's Reception: 10 p.m., Monday, August 22, Trocadero, George Street.

Plenary Sessions and Lectures: University of Sydney.

Congress Dinner: 7 p.m., Wednesday, August 24, David Jones, Limited, Elizabeth Street.

Henry Simpson Newland Oration: 8.30 p.m., Thursday, August 25, Great Hall, University of Sydney.

Congress Ball: 9 p.m., Friday, August 26, Trocadero, George Street.

Accommodation.

It is appreciated that many members will make their own arrangements for accommodation while in Sydney, and it is not the policy of the Executive Committee to control or direct this. However, a subcommittee has made liaison with the principal hotels *et cetera*, and any member who wishes his or her accommodation arranged via this channel is invited to apply to the Honorary General Secretary of Congress.

Sponsoring.

As on previous occasions, arrangements have been made to sponsor those from other States who desire it. A number of local members has offered to sponsor visitors—that is, to contact them on arrival, to guide them to their hotels and to act as general advisers and persons of reference for information while the visitor is in Sydney. Those who wish to be sponsored are cordially invited to notify the Honorary General Secretary of Congress.

Transport: Members' Own Cars.

Arrangements have been made with the Traffic Department of the New South Wales Police for the issue of "stickers" to all members on registration. These will be affixed to the windscreen and are expected to facilitate the control and parking of cars.

Visiting members are advised that overnight and street parking is difficult in Sydney and that it might be better to rely on private, taxi or hire car services. Visitors are also advised that travel during peak hours is apt to be tedious and uncertain. Accordingly, ample time should be allowed to ensure punctuality at functions. A special parking area will be available in the University grounds in front of the Old Medical School.

Registration.

All members are required to register. Visiting members should do this as soon as possible after arrival in Sydney, and local members as soon as possible after the office opens. The Registration Office will be in the Union Hall, University of Sydney. The Registration Office will be open as follows:

Saturday, August 20, 9 a.m. to 12 noon.

Monday, August 22, 9 a.m. to 5 p.m.

Tuesday, August 23, to Friday, August 26, 9.30 a.m. to 5 p.m.

Badges.

Each member will be issued with a bronze badge on registration together with a lady's oxidized silver badge. It is requested that these badges be worn at all Congress gatherings.

Programme et cetera.

On registration, each member will receive a folder containing a copy of the Congress Handbook, the Scientific Programme and the Handbook of Entertainment and Amenities, a map of Sydney (by courtesy of Burroughs Wellcome, Limited), and certain other items. No copies of the programme will be distributed before registration. The scientific programme follows the usual plan, and consists of five plenary sessions and sectional meetings in all branches of medicine. Details have already been published in the issues of July 9 and 16, 1955.

The plenary sessions and sectional meetings will be held in the Great Hall and various lecture theatres in the University of Sydney. Full details will be included in the programmes to be issued on registration.

Meetings have been planned to commence at 9.30 a.m. and end at approximately 4.30 p.m., excepting Tuesday, when the plenary sessions will end at about 5.15 p.m. The luncheon interval will be from 12.30 to 2 p.m. It will not be easy to go into the city for lunch and to return in time for the afternoon meetings, and members are advised that arrangements have been made for them to obtain a cafeteria lunch at the University Union and at Manning House.

Transport to and from the University.

To assist members, arrangements have been completed with the New South Wales Transport Department to run a special bus service from the city to the University. This will depart from the centre of Martin Place, between Pitt and Castlereagh Streets, at 8.55 a.m., from Tuesday to Friday inclusive. The return services will depart from the University in front of the Great Hall at 5.20 p.m. on Tuesday, August 23, and 4.30 p.m. on Wednesday, August 24, to Friday, August 26. This service will be free to members of Congress and should prove very convenient.

Church Services.

Official Congress church services will be held as follows:

Church of England: St. Andrew's Cathedral, 11 a.m., Sunday, August 21.

Combined Presbyterian and Methodist: Assembly Hall, 11 a.m., Sunday, August 21.

Roman Catholic: St. Mary's Cathedral, 11 a.m., Sunday, August 21.

Jewish: The Great Synagogue, 9.30 a.m., Saturday, August 20.

Members attending these services are requested to wear academic robes, to assemble outside the churches, and to enter in procession. Special accommodation will be reserved for wives of members.

The Jewish service will be followed by a reception.

Official Opening.

His Excellency the Governor-General of Australia has graciously accepted an invitation to open Congress officially in the Sydney Town Hall on Monday, August 22. *Members should be seated by 8.15 p.m.* An organ recital will be held from 7.45 p.m. to 8.30 p.m. Following the official opening, the presidential address will be delivered by Sir Charles Bickerton Blackburn.

Subsequently, those attending this function will be received by the President at a reception. As facilities for this are not available in the Town Hall, the reception will be at the Trocadero, which is adjacent. Following the reception, supper will be served. Invitation and entrée cards will be issued to members on registration. Dress will be evening dress with academic robes and decorations.

Congress Dinner.

The Congress Dinner will be held at David Jones, Limited (Elizabeth Street entrance), on Wednesday, August 24. His Excellency the Governor of New South Wales has graciously accepted an invitation to be present.

Members and their wives may attend this function, and as numbers have to be restricted, early application is advisable. The tickets are £3 10s. per person. The time will be 7 p.m. for 7.45 p.m. Dress will be evening dress with decorations. In view of the parking difficulties members are advised to arrive early.

Henry Simpson Newland Oration and Conferring of Honorary Degree.

A combined function will be held in the Great Hall of the University on Thursday, August 25, at 8.30 p.m. The University of Sydney has advised that an honorary degree will be conferred on Dr. Louis H. Bauer, Secretary-General of the World Medical Association. The representative of the Parent Body, Dr. A. Talbot Rogers, will present the Gold Medal of the Association to Sir Henry Newland in recognition of his outstanding services. The Henry Simpson Newland Prize in Surgery will be presented to the winner of the prize essay.

The second Henry Simpson Newland Oration will then be delivered by Dr. Louis Bauer.

Dress will be evening dress with academic robes.

Congress Ball.

Following the usual custom, the members of the New South Wales Branch will be the hosts at the Congress Ball, which will be held at the Trocadero on Friday, August 26, at 9 p.m. All visiting members and their wives are invited to attend. Local members who seek information about this function are referred to the Medical Secretary, New South Wales Branch. Dress will be evening dress.

Social Entertainment.

A comprehensive programme has been arranged for the entertainment of members and their wives. The details will be included in the Social Programme to be issued on registration.

It is the policy of the Executive Committee that formal invitation cards will be issued to all members attending parties. Through mutual friendships, some members will receive such invitations from hosts and hostesses in advance, but all members are requested to call personally, or have their wives call, at the Social Club as soon as possible after arrival in Sydney to avail themselves of the hospitality which their colleagues here are anxious and happy to extend.

The Social Club will be open as follows:

Friday, August 19, 9.30 a.m. to 5 p.m.

Saturday, August 20: 9.30 a.m. to 1 p.m.

Monday, August 22: 9 a.m. to 5 p.m.

Tuesday to Friday, August 23 to 26: 9.30 a.m. to 5 p.m.

Hosts and hostesses are extending hospitality at parties and excursions from Saturday, August 20, onwards, so please report early to the Social Club. In arranging the social programme, the policy has been to restrict parties to the central area because of time factors and the difficulties of transportation in heavy traffic. In general, the dress for private parties in the evening will be the same as that for the official functions to follow. This will save members changing between such parties and subsequent functions.

A special programme has been prepared for the entertainment of juniors who accompany members. The

organizers of this section would appreciate any information members could supply in advance through the Honorary General Secretary of Congress.

Dress.

Dress orders for the various official occasions will be shown in the programme. Members are advised that evening dress will be the usual order for evening functions and that they should bring this in preference to dinner jackets. Members are requested to bring their academic robes and decorations.

Garden Party, Callan Park Mental Hospital.

The Honourable M. O'Sullivan, M.L.A., Minister for Health, has kindly arranged for a garden party to be held in the grounds of Callan Park Mental Hospital, Leichhardt, at 3 p.m. to 5 p.m. on Monday, August 22.

Hobbies.

An interesting exhibition of doctors' hobbies is being arranged. Members who desire to participate are invited to notify the Honorary General Secretary of Congress.

Trades Exhibition.

A large Trades Exhibition has been arranged and will allow members to see some of the products of a number of local and overseas manufacturers. It will be open from Saturday, August 20, to Friday, August 26, inclusive, and all members are asked to make a special effort to visit this exhibition at some time during Congress, and if possible to attend the official opening. The exhibition will be officially opened by the President, Sir Charles Bickerton Blackburn, at 9.30 a.m. on Monday, August 22, in the Peter Nicol Russell School of Engineering Lecture Theatre.

Apart from this and the official opening of the Hobbies and Scientific Exhibitions, there will be no official functions on Monday, August 22, until the inaugural meeting at the Town Hall at 8.30 p.m.

Scientific Film Programme.

Associated with the Trades Exhibition there will be a showing of scientific films throughout the whole week of Congress. Details of the showing will be published in the *Scientific Handbook*.

Museum and Scientific Exhibition.

An interesting and comprehensive *Museum and Scientific* Exhibition will be displayed in the drawing room of the Department of Electrical Engineering.

Sports.

The *sports programme includes golf competitions* for men and women, as well as facilities for participating in golf, racing, bowls and game fishing. Those interested are asked to notify the Honorary General Secretary of Congress.

Crèche.

Mrs. Douglas Anderson, 4 Highview Avenue, Neutral Bay, has offered to provide at her home facilities to mind, under trained supervision, the children, up to the age of nine years, accompanying country and interstate visitors to Congress. This service will be available day and evening. Children will be picked up and returned to the Social Club. Mrs. Anderson would be glad of three weeks' notice.

Information Sought: A Request.

To assist the organizers, and especially for the arrangement of entertainment, the Honorary General Secretary requests that members of Congress kindly reply as early as possible, supplying the following information (if they have not already done so): (i) Date and time of arrival in Sydney. (ii) If they will be attending the dinner and if their wives will be attending also. (iii) Information *re* any juniors who will be coming with them.

Corrigendum.

AN error appears in the article entitled "Rh Antibodies", by Vera I. Krieger, D.Sc., published in the issue of July 23, 1955. On page 121, at the end of the ninth line, "50%" should read "only 5%". The complete sentence will then

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 16, 1955.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	3	4(2)	4(1)	..	1	..	..	..	12
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	9(9)	6(1)	..	..	..	..	..	15
Diphtheria	..	6(6)	6(4)	..	8(8)	..	..	..	20
Dysentery (Bacillary)	..	2(2)	3(3)	..	..	..	..	..	5
Encephalitis	..	1	..	1	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	45(8)	80(31)	..	10(7)	9(5)	..	1	..	145
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	1	..	1
Leptospirosis	1	..	2	..	..	..	..	..	3
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	3(2)	..	3	..	..	1	..	..	7
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	2(1)	3(1)	1	2(1)	..	..	..	..	8
Puerperal Fever	..	..	1	..	..	..	..	..	1
Rubella	..	28(21)	..	..	4(3)	..	..	..	32
Salmonella Infection	..	..	..	..	1(1)	..	..	..	1
Scarlet Fever	7(5)	17(11)	13(2)	3(3)	1(1)	1	..	..	42
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	2	..	..	..	..	..	..	2
Trachoma	..	..	..	..	6(2)	..	..	..	6
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	41(22)	18(11)	17(12)	2(2)	11(10)	3(1)	..	..	92
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	1(1)	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

read as follows: "In a series of 200 tests we found identical titres by both methods in 30%, a higher titre in the indirect Coombs test in only 5%, but a higher albumin titre in 65%." We regret this error.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Annual Subscription Course.

THE following lectures will be given by Sir Stanford Cade and Professor Brian Windeyer during the second week of their visit to New South Wales:

Monday, August 8, 8.15 p.m., at the Stawell Hall, 145 Macquarie Street, Sydney, in conjunction with the College of Radiologists of Australasia, New South Wales Branch: "Skeletal Metastases", Professor Windeyer.

Tuesday, August 9, 8.15 p.m., at the Stawell Hall: "Reticuloses", Professor Windeyer.

Wednesday, August 10, 8.15 p.m., at the Robert H. Todd Assembly Hall, 135 Macquarie Street, Sydney, in conjunction with the Urological Society of Australasia: "Carcinoma of Penis and Testis", a combined lecture.

Thursday, August 11, at 8.15 p.m., at the Robert H. Todd Assembly Hall: "Carcinoma of the Breast", a combined lecture.

For further information, apply to The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8.

Notice.

VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

Section of Preventive Medicine.

THE next meeting of the Section of Preventive Medicine of the Victorian Branch of the British Medical Association will be held in the Medical Society Hall, 426 Albert Street, East Melbourne, on Thursday, August 11, 1955, at 4.30 p.m. Associate Professor F. Duras, Director of Physical Education, University of Melbourne, will deliver an address entitled "The Medical Profession and Sex Education". This address will be illustrated by a sound film on the subject. All members of the Branch are invited to be present.

University Intelligence.

UNIVERSITY OF ADELAIDE.

The Shorney Prize.

THE Shorney Prize for 1954 has been awarded to Dr. W. E. Fleming, of Melbourne, for a thesis entitled "Chronic Osteitis of the Maxilla in its Relationship to Maxillary Sinusitis". This prize, established for the purpose of perpetuating the memory of the late Herbert Frank Shorney, lecturer in ophthalmology in the University of Adelaide from 1926 to 1933, is offered periodically. It is awarded to the candidate who in the opinion of the examiners has made the most substantial contribution to knowledge of the subjects of ophthalmology or diseases of the ear, nose and throat. It is offered alternately for work in ophthalmology and in diseases of the ear, nose and throat.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Roberts, John, M.B., B.S., 1954 (Univ. Sydney), 98 Church Street, Newcastle, New South Wales.

The undermentioned have applied for election as members of the Victorian Branch of the British Medical Association:

Willington, Clayton Louis, M.B., B.S., 1955 (Univ. Adelaide), c.o. Gippsland Base Hospital, Sale, Victoria.

Sladdin, John Naulty, M.B., B.S., 1952 (Univ. Adelaide), Box 82, Macarthur, Victoria.

Deaths.

THE following deaths have been announced:

MURRAY.—Alexander Francis Murray, on July 7, 1955, at sea.

OLD.—George Greensill Old, on July 22, 1955, at Sydney.

Diary for the Month.

AUG. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 12.—Tasmanian Branch, B.M.A.: Branch Council.
AUG. 15.—Victorian Branch, B.M.A.: Finance Subcommittee.
AUG. 16.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 17.—Western Australian Branch, B.M.A.: General Meeting.
AUG. 18.—Victorian Branch, B.M.A.: Executive of Branch Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.